SERIES 3, Vol. 23, No. 1 JANUARY, 1940

AMERICAN JOURNAL OF OPHTHALMOLOGY

CONTENTS

DEPARTMENTS

For complete table of contents see advertising page V

Subscription price in United States ten dollars yearly. In Canada and foreign countries twelve dollars. Published monthly by the George Banta Publishing Company, 450 Ahnaip Street, Menasha, Wisconsin, for the Ophthalmic Publishing Company. Subscription and Editorial Office: 640 S. Kingshighway, Saint Louis, Missouri. Entered as second class matter at the post office at Menasha, Wisconsin.

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 23 JANUARY, 1940 NUMBER 1

THE PATHOLOGY AND PATHOGENESIS OF SYPHILITIC PRIMARY OPTIC ATROPHY

A CRITICAL REVIEW*

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In 1932, one of us (J. E. M.) published a review of the literature on syphilitic primary optic atrophy,[1] with a bibliography of 96 references. A few months later a somewhat similar review appeared by Schiff-Wertheimer and L'Hermitte,[2] with a still more extensive bibliography of 271 references, dating from 1904.[3] These two reviews considered mainly the pathogenesis and treatment of optic atrophy, and were in general agreement on most points.

THE PATHOLOGY OF OPTIC ATROPHY

It was pointed out that while the pathology of optic atrophy was fairly well known, there were many gaps in our knowledge. There is general agreement among pathologists that the process begins in the intracranial portion of the optic nerve distal to the chiasm; that the degeneration occurs first in the marginal fibers of the nerve; and that there is thickening and perivascular round-cell infiltration of the overlying membranes, especially the pia, and of the connective-tissue septa of the nerve. There is, however, no agreement as to the important question of the relationship, if any, of the inflammatory changes in the membranes and septa to the degenerative process in the nerve, since the former are frequently found in neurosyphilitics who do not show the latter.

It was clear in 1932, as both of these lengthy reviews pointed out, that the development of optic atrophy probably does not depend on the actual presence of *T. pallida* in the optic nerve or visual pathways, since the organism, although demonstrable in one of these locations in a small proportion of patients with optic atrophy, can be likewise so demonstrated in patients without optic atrophy.

In Moore's 1932 review, it was pointed out that, among others, Wagner-Jauregg[4] objected to the validity of most of the pathologic work so far available on syphilitic optic atrophy on the ground that the majority of the patients studied at necropsy were paretics or tabo-paretics in whom the widespread pathologic changes in the brain rendered unsafe any conclusions as to such a highly specialized portion as the visual tracts. As Wagner-Jauregg emphasized, the essential need of pathologic study of pure tabes dorsalis with optic atrophy, and particularly with beginning optic atrophy, had not been satisfied.

Since 1932, nothing of importance has been added to the literature which bears directly on the pathology of syphilitic optic atrophy. Biffis[5] has studied the optic

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This article is appearing simultaneously in the American Journal of Syphilis, Gonorrhea and Venereal Diseases.

tracts of 12 patients with paresis, of which 2 showed histologic evidence of optic atrophy, but his paper contributes nothing new. Malbrán[6] and Krainer[7] have contributed little or nothing bearing on the development of optic atrophy. Behr[8] has published a paper on the anatomy of the connective-tissue septa of the optic nerve and the blood supply of the optic pathways that is of interest in connection with the vascular theory of the pathogenesis of optic atrophy to be referred to later.

In a still later paper, Behr[9] believes that it is possible to differentiate between genuine tabetic atrophy of the optic nerves, as a quaternary form of syphilis, and a pseudotabetic atrophy as a tertiary form. Only the optic nerve provides for the relative arrangement of nerve fibers and blood vessels in the connective-tissue-like, hollow cylinders (septa). Moreover, the optic nerves, like the remaining mass of cerebral substance, possess a glial limiting membrane, where mesoderm comes in contact with ectoderm, that is responsible for the selection of available food for the ganglion cells and nerve fibers. Therefore, Behr believes that in the optic nerves the food issuing from the vessels collects in the hollow spaces of the septal framework, and only the function of the surrounding glial membrane releases the blood little by little. A disturbance in the region of the glial limiting membranes must, it follows, endanger the nutrition of the nerve fibers.

Syphilis in the central nervous system takes two principal forms, says Behr: a genuine syphilitic inflammation and a metasyphilitic or parasyphilitic degeneration. The first comes principally from the mesoderm (in the optic nerves, the pia, and septa), while the second represents a progressive degeneration of the nerve substance. There are, accordingly, only two forms of syphilitic disease of the optic nerves: (1) genuine acute or chronic syphilitic inflammation, issuing from the mesoderm, with secondary degeneration of the nerve fibers; and (2) primary degeneration, in the form of tabetic (whether or not associated with paresis) atrophy of the optic nerves.

Syphilitic inflammations develop in the optic nerves as: (1) meningeal, (2) vascular, and (3) a combination of the two. From the inflammation of the pia the septal system is involved, becoming edematous, thick, and strangling the bundles of nerve fibers. The resulting disturbances in function consist of a decrease in central vision and concentric visual-field defects. In this type, if the diagnosis is made early enough and energetic antisyphilitic treatment is given, complete healing may occur. On the other hand, the inflammatory granulation tissue may heal with scarring, resulting in permanent, serious loss of function. Vascular involvement, with the intrusion of inflammatory proliferation into the nerve parenchyma along the blood vessels that enter from the pial coat, is less common, Behr thinks. When it occurs, the nerve fibers are the victims of ischemia; peripheral visual-field constrictions may terminate in hemianopsia. There is still no reliable anatomical evidence of syphilis of the vessels of the optic nerves.

Fifty optic nerves of tabetic patients with and without visual disturbances have been pathologically examined in Behr's clinic. The atrophic process begins in the peripheral septal section; that is, between the eye and the chiasma. From this region the degeneration descends and ascends. If treponemes can be demonstrated at all, they are found only in the supporting structure of connective tissue but never in the nerve mass. Behr thinks that the increasing difficulty in the

movement of the lymph stream that nourishes the nerve-fiber bundles produces degeneration.

There are three basic types of further development of tabetic atrophy, with many transitional forms: (1) The disease proceeds from the periphery, concentrically towards the axis. In this type is found clinically concentric constriction of the visual fields. Color and central vision remain intact for a long time. Dark adaptation does not suffer. (2) The disease may proceed from the pia in wedge-shaped projections. Here the fields show sector defects both for form and color. Dark adaptation and central vision remain intact for a long time. (3) A cross section of the nerve shows slight degeneration of all the fibers. Clinical study of this type shows that the peripheral visual field remains almost normal, although central vision soon decreases. Dark adaptation is also early reduced. In Behr's opinion, this last form, but not the other two, is made worse by antisyphilitic treatment.

The pathogenesis of optic atrophy

It is an old observation that primary optic atrophy in neurosyphilis occurs with greatest frequency, if not almost exclusively, in tabes dorsalis; that in many instances it develops before definite evidence of posterior-column damage is obvious (the optic atrophy of "pre-ataxic" tabes); and that it may occur in occasional cases of otherwise apparently stationary tabes, with negative serologic findings in blood and spinal fluid. Especially noteworthy is the fact that optic atrophy is rare in uncomplicated paresis, even though widespread meningovascular and parenchymal changes in other parts of the brain are always present; but it is very common in taboparesis.

In view of the pre-ataxic occurrence of optic atrophy in neurosyphilis, that is, the presence of optic atrophy in patients who show no evidence whatever of posterior-column damage at the time of development of the optic atrophy or later, many ophthalmologists and neurologists have felt, on clinical rather than pathologic grounds, that optic atrophy might be due to basilar meningitis rather than to tabes; and indeed that the optic atrophy of basilar meningitis carried a more favorable prognosis than did the tabetic form. These suggestions, discussed by Moore[1] in 1932, are of particular interest in view of the newer conceptions of optochiasmic arachnoiditis, presently to be discussed.

If basilar meningitis is shown to be a common finding in syphilitic primary optic atrophy, tabetic or otherwise, the pathogenesis of optic atrophy is readily understood, on the simple ground of constriction of the optic nerve itself, or its blood supply, or both. However, since basilar meningitis in tabes is rarely extensive (Richter[10]), this facile explanation does not suffice for the vast majority of cases of primary optic atrophy.

Indeed, to understand the pathogenesis of primary optic atrophy, one must understand, as Moore[1] and Schiff-Wertheimer and L'Hermitte[2] pointed out in 1932, the pathogenesis of tabes dorsalis itself. This is as yet far from clear. Tabes differs in important clinical and pathologic respects from all other types of neurosyphilis. These differences may be briefly summarized as follows:

(1) Its longer incubation period. The average interval from infection to the development of untreated meningovascular neurosyphilis is 14 years; of paresis, 19 years; and of tabes dorsalis 24 years (Hopkins[11]).

(2) The peculiar selectivity of tabes for the posterior columns of the spinal

cord and its strict limitation in the cerebrum to four isolated disturbances; namely, (a) the pupillary changes of anisocoria, miosis, and abolition of the light reaction, (b) disturbances of sensation in the distribution of the fifth cranial nerve, (c) oculomotor palsies, and (d) primary optic atrophy. Diffuse meningovascular neurosyphilis and paresis exercise no such selectivity.

(3) The relative refractoriness of tabes to specific or nonspecific antisyphilitic treatment, as compared with other types of neurosyphilis.

(4) The fact that the reagin content of both blood and spinal fluid is low in tabes as compared to other types of neurosyphilis. With serologic tests of average sensitivity, the blood is negative in about 30 percent of frank tabetics; the spinal fluid negative (as to reagin content) in about 20 percent, and when positive usually only weakly so.

(5) Most important of all, the lesions of tabes are remarkable for the absence, rather than the presence, of the causative organism of syphilis, the *T. pallidum*. In both early and late meningeal or diffuse meningovascular neurosyphilis, and in paresis, the organisms can usually be demonstrated without difficulty and in large numbers. In tabes, on the other hand, and in spite of many attempts by neuropathologists the world over, organisms have been found in only eight cases by five different observers (Noguchi, Versé, Jahnel, Richter, and Igersheimer[12]). In only one case has the *T. pallidum* been found in the visual pathways of a tabetic patient (Igersheimer); the other reports of positive findings are all in patients with paresis.

These striking differences between tabes and other types of neurosyphilis have led to many theories regarding its pathogenesis (best summarized for an English-reading group by Hassin[13] and Stern[14]), none of which are as yet acceptable. As for tabes, so also for its common manifestation, primary optic atrophy, the best that can be said is that its cause is still unknown.

New theories of the pathogenesis of optic atrophy

Since the publication of the two long 1932 reviews, five new theories of the pathogenesis of tabes dorsalis or of optic atrophy have gradually evolved. These are: (1) that tabes may be due to the coexistence of syphilis and of lymphogranuloma inguinale; (2) that optic atrophy depends on a nutritional disturbance of the optic nerve due to syphilitic involvement of the blood supply of the nerve; (3) that optic atrophy depends on a disturbance of the relationships between systemic blood pressure, retinal blood pressure, and intraocular tension with resultant disturbance in the nutrition of the optic nerves or retina; (4) that optic atrophy depends on meningeal inflammation with the development of optochiasmic arachnoiditis, which constricts the nerve or its blood supply; and (5) that optic atrophy may be due to a combination of nutritional (vitamin) deficiency and neurosyphilis.

Each of these theories will be discussed in detail, together with such data as to the treatment of syphilitic primary optic atrophy as are pertinent to the argument. Since at least three of these theories utilize relatively new methods of treatment as vital elements in their development, it is therefore necessary, in order to evaluate the theories and the results of the new therapeutic procedures, to consider afresh the course of untreated primary optic atrophy. To be acceptable, either in proof of a theory or as a practical measure, any treatment proposed must naturally improve on the results without treatment.

The course of untreated primary optic atrophy

The rate of progression of syphilitic primary optic atrophy from onset to blindness is extremely variable. The older literature (Behr,[15] Stargardt,[16] Igersheimer[17]) contains numerous statements to the effect that the duration of the process may vary from as short a time as a few weeks to as long as 12 years. Two recent writers, Heim[18] and Miklos,[19] suggest that the course of the disease may not be so rapidly progressive as in former years. Heim reports one patient in whom optic atrophy was diagnosed in 1898 on the basis of pale discs, though visual acuity and visual fields were normal, in whom there was no visual failure for 16 years under a treatment régime limited to the administration of potassium iodide by mouth. In 1914, two injections of neoarsphenamine were given which caused prompt partial visual failure and led to the abandonment of all treatment. Heim records five other cases of optic atrophy under constant observation for 5 years, during which they received no other treatment than certain proprietary drugs called iodolecithin or sajodin (an iodine preparation) in combination with tetrophan. No progression of the atrophy occurred.

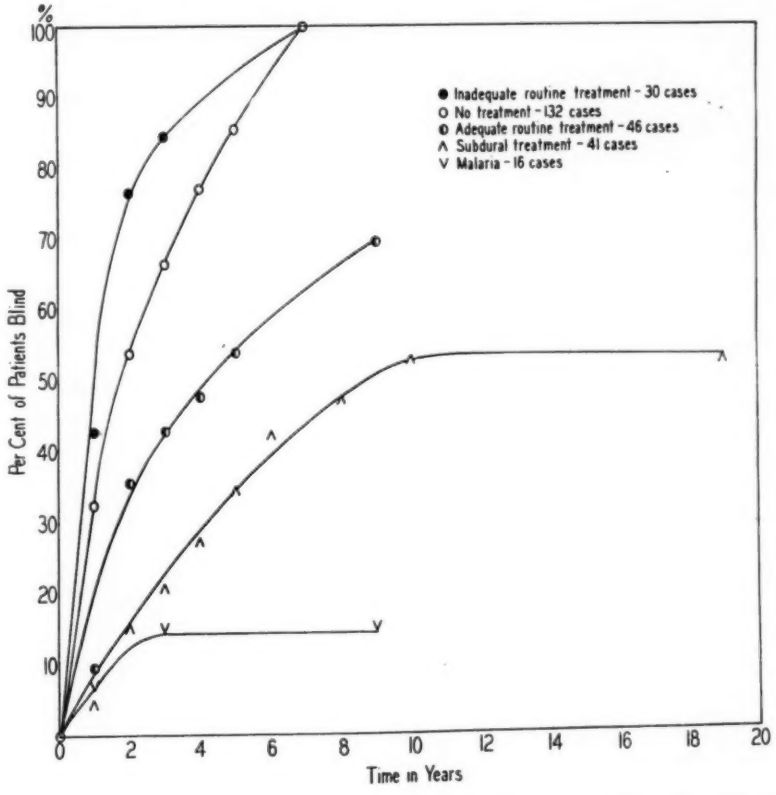


Fig. 1. The average interval from onset of symptoms to blindness in patients with syphilitic primary optic atrophy untreated and treated by various methods (from Moore, Woods, Hopkins, and Sloan[21]).

Among Miklos's 17 carefully studied cases, there was one patient who had

either arrested or slowly progressive optic atrophy for 5 years, two for 4 years, one for 3 years, and two for 2 years before antisyphilitic treatment was begun. He believes that such slowly progressive cases are more frequent than formerly, and this may be due to a general amelioration in the course of syphilis influenced either by decrease in the virulence of the treponeme or the gradual immunization of the human race.

Such cases are, however, the exception rather than the rule. It is a remarkable fact that only within the last year has there become available accurate statistical information as to the usual duration of optic atrophy from onset of symptoms to blindness in untreated patients. These data are continued in reports by Gross and Lehrfeld[20] of 117 untreated patients from the Wills Hospital (Philadelphia), and in those by Moore, Woods, Hopkins, and Sloan,[21] of 132 untreated cases. In these two series, aggregating 249 untreated patients, 70 percent were blind within 3 years, 90 percent within 5 years, and all were blind within 9 years (in the Johns Hopkins material, see figure 1, all were blind within 7 years). A course more slowly progressive than this, as described by Heim and Miklos, must therefore be extraordinarily rare.

It is clear, therefore, that before any treatment method can be described as successful, a minimum observation period of at least 3 years is essential.

The relationship of tabes to lymphogranuloma inguinale

This theory need not detain us long, since it has no relationship to optic atrophy. Several groups of workers, impressed by the peculiarities of tabes described above, have studied the possibility that tabes might, at least in some instances, be due to the association of syphilis with lymphogranuloma inguinale. This hypothesis was prompted by the fact that both diseases are transmitted in the same manner and that the virus of lymphogranuloma is known to produce a meningo-encephalitis in experimental animals and rarely in man.

Levaditi and his collaborators,[22, 23] Lacassagne and Lebeuf,[24] Ionesco-Mihaesti and co-workers,[25, 26] and Videla and Pastor[27] have approached the problem by performing Frei tests (supposedly specific for lymphogranuloma) on patients with tabes, with conflicting results; or by attempting to produce a tabeslike disease in animals by inoculation with the virus of lymphogranuloma, also with conflicting results.

So careful a student of neurosyphilis as Jahnel[28] believes that this hypothesis has no justification in fact. It is quite clear that optic atrophy does not develop in patients with lymphogranuloma alone; or even frequently in those patients in whom this disease is most often associated with syphilis (Negro females).

The theory of nutritional disturbance of the optic nerve due to syphilitic lesions of its blood vessels

Abadie has been a student of optic atrophy in general, rather than of syphilitic optic atrophy in particular. In a series of communications,[29–35] he suggested that in many cases of tabetic optic atrophy the retinal arteries appeared on ophthalmoscopic examination to be constricted. He hypothesized that this constriction was due to a disturbance of the sympathetic nervous control of the central artery of the retina, brought about in syphilis, at least, by a lesion in or near the ciliospinal center in the medulla. He believed the lesion comparable to that which produces the Argyll-Robertson phenomenon. The constriction of the vessels was, he thought, progressive and

permanent, leading to progressive ischemia of the optic nerve and retina and consequent ascending degeneration of the optic nerve.

Abadie's theory was completely undocumented by pathologic or indeed by clinical evidence, but it attracted the attention of other observers, who developed it with variations. The general idea was that the degeneration of the nerve fibers was due to ischemia produced by spasm of the arterial blood-vessel walls by syphilitic inflammatory tissue or by involvement of their sympatheic nerve supply.

As Schiff-Wertheimer and L'Hermitte[2] pointed out, however, there is absolutely no pathologic background for this theory, since in syphilitic primary optic atrophy vascular lesions in the optic nerve and retina are usually lacking; when they do occur they may conceivably be the result, rather than the cause, of the nerve degeneration. In other conditions in which vascular lesions are actually present, for example in retinal arteriosclerosis, even including changes in the central artery, optic atrophy is comparatively rare.

Nevertheless, and purely empirically, Abadie suggested the treatment of optic atrophy by means of various vasodilator drugs. With complete disregard of physiology and pharmacology, it was at first recommended that these be administered by retrobulbar injection, of course with the idea that the direct deposit of an atropine solution close to the central artery of the retina would have a greater vasodilator effect than if it was administered by some other route. Subsequently, Abadie himself agreed that the vasodilator effect of atropine upon the retinal vessels, if any, was as great when the drug was administered by the subcutaneous or intravenous as by the retrobulbar route; and still other ophthalmologic enthusiasts think that it is equally effective by mouth.

Abadie advised atropine treatment for tabètic optic atrophy as well as for other nonsyphilitic types. In syphilitic patients, he combined daily subcutaneous injections of atropine with daily intravenous injections of mercury cyanide, both continued indefinitely. However, in none of his many published papers is there any adequate documentation of the value of the method in syphilitic primary optic atrophy. In his last few papers, published shortly before his death in 1932, he advised acetylocholine for the same purpose, to relieve spasm of the retinal vessel, but again does not document.

There is disagreement in the literature as to the physiologic effect of atropine, administered by any route, upon the visible retinal blood vessels. Schiff-Wertheimer and L'Hermitte[2] say that there is no visible dilatation of the retinal blood vessels after its use, nor will it overcome the retinal vascular spasm caused by the intravitreous injection of adrenalin. Vele,[36] on the other hand, claims that when atropine is given subcutaneously, there is visible dilatation of the retinal vessels and hyperemia of the optic nerve. Most of the ophthalmologists who have used the method in the treatment of optic atrophy omit mention even of this elementary point.

Atropine by some route of administration, or other vasodilator drugs such as acetylocholine, have been used in the treatment of various types of optic atrophy by a number of investigators (Samkowskij,[37] Abbasov,[38] Maslenikov,[39] Springovitsch,[40] Bursuk,[41] Mariotte and Lugli,[42] Gapeeff,[43] Kazlauskas,[44] Stegemann,[45] Simko,[46] Kosjmin,[47] Vele,[48] Prister,[49] Fejer,[50] Folk,[51] and Oniki[52]). On the whole, all these papers are utterly unconvincing. All types of optic atrophy are grouped together, and there is usually

no means of telling from the published papers as to how many of the patients had syphilis, or, if these data are given, what were the results in the syphilitic as compared to the nonsyphilitic groups. In the case of syphilitic patients, most authors administered concomitant antisyphilitic treatment as well as the vasodilator drug. Finally, in practically all instances, the follow-up period of observation is so short that the results, if any, are wholly uncertain.

In spite of these defects, however, it may be said that apparently this method of treatment is of little or no value in the treatment of tabetic optic atrophy, as exemplified by the papers of Simko, Prister, Fejer (23 cases of tabetic optic atrophy), Springovitsch (31 cases of syphilitic optic atrophy), Kazlauskas, Kosjmin, and our own personal experiences. If improvement was noted at all in these cases, it was usually of very short duration (days or weeks only) to be followed by continued downhill progress. On the other hand, a number of authors agree (Springovitsch, Kosjmin, Prister, Vele) that vasodilator drugs may often produce striking and permanent improvement in other types of optic atrophy, especially those due to retrobulbar neuritis or to unknown causes.

On the whole, the theory is one which seems, at least as to syphilitic primary optic atrophy, unsupported by any known anatomo-pathologic facts, and the rationale of treatment founded on unsupported supposition and lacking all adequate documentation.

Acting on the same theory, that optic atrophy was due to vasoconstricting spasm of the blood vessels of nerve and retina, Magitot,[53] Löwenstein,[54] and Nižetić, Spiridonović, and Bukarov[55] have proposed carotid sympathectomy as a permanent method of vasodilatation. Magitot's two papers refer to the same six patients, of whom only two had neurosyphilis. There are interesting physiologic effects of the operation on retinal and systemic arterial blood pressure, all of which are apparently transitory (lasting a few days or weeks). Nižetić and his co-workers have operated on 32 patients with supposed syphilitic optic atrophy, of whom 20 were unchanged, 7 improved, and 5 worse over observation periods ranging from a few days to several months. Blobner[56] has operated on 13 patients with either tabetic optic atrophy or pigmentary degeneration of the retina, with entirely negative results, and has abandoned the procedure. Though some patients so treated are said by all the investigators named to have improved, the ophthalmologic and neurologic details are, to say the least, sketchy and the period of postoperative observation very short (in no case longer than one year, and in one syphilitic patient six days!).

This is perhaps the best place to notice the papers of Hamburg,[57] whose curious theory seems to belong with the theory of disturbance of nutrition of nerve fibers from faulty blood supply. Hamburg thinks that there is a similarity between the optic atrophy of tabes and the toxic amblyopia due to methyl alcohol. It has been suggested that the latter is due to decreased consumption of oxygen by body cells. Hamburg postulates that perhaps cellular metabolism is slowed in the optic nerves of some tabetics, and adduces as evidence for this hypothesis that in tabetic atrophy, visual acuity is often better in the morning than in the evening (this due to the expenditure of oxygen by work and its conservation during rest). He suggests that cellular metabolism and oxygen utilization may be increased by the intravenous administration of 1 mg. of thyroxine every day or every other day, combined with the intra-

muscular administration of potassium permanganate 1 mg. daily. By this routine he has treated 35 patients with tabetic optic atrophy, the majority of whom improved, and some of them remarkably (for example, visual acuity increased in one case from 3/50 to 3/12, in another from 3/25 to 3/10), but he does not state how long the improvement lasted nor for how long the patients were followed. The observation periods were obviously very short.

Löffler[58] made partial use of Hamburg's method by treating 16 patients three times weekly with 1.0 c.c. of 0.25-percent solution of potassium permanganate intramuscularly. These patients were observed for from one-half to three years. One can draw no definite conclusions, because many of them had routine antisyphilitic treatment or malaria as well as the permanganate. During the observation period, two went blind, six remained unchanged, and eight became worse. There is obviously nothing impressive about these figures.

Another unusual method of treatment, whose rationale, if any, seems to depend on increasing the blood supply of the optic nerve and retina, is advocated by Saitzew.[59] Because placental blood is so rich in hormones and is so biologically active, he conceived the idea that it might exert a beneficial effect on the course of tabes and of tabetic atrophy. Over a period of three-and-a-half years he has given it to 14 patients with optic atrophy. Fifteen to 20 subcutaneous injections of 6 to 10 c.c. every day or every other day were given. Six of the patients were blind before treatment. In the remaining eight the vision before treatment was reduced to between 0.5 and 0.01. After the treatment, three are said to have been subjectively better, four objectively better, and one unchanged.

In summary, it may be fairly said that these various attempts to dilate the intraocular vessels and thereby influence the course of optic atrophy are so open to criticism in both their conception and execution that they may be dismissed without further consideration.

The Theory of the Interrelationship of Systemic Blood Pressure, Retinal Blood Pressure, and Intraocular Tension

In 1917, Bailliart[60] first reported the results of dynamometric studies of the retinal circulation, and in 1923 gathered together his observations in a monograph. The intraocular tension and retinal vascular pressure are measured by an instrument known as an ophthalmodynamometer. This is applied directly to the sclera, and is so calibrated that the several pressures may be read on a scale. By means of direct ophthalmoscopic observation of the fundus, both systolic and diastolic arterial and venous pressures may thus be directly measured in the retina. The systolic retinal arterial pressure is considered to be the point at which the retinal arteries emerging from the disc are completely obliterated by pressure; the diastolic arterial pressure, the point at which maximum pulsations of the retinal arteries appear. The venous systolic pressure is likewise the point at which the central retinal veins are completely obliterated by pressure; the venous diastolic pressure, the point at which pulsation in the retinal veins first appears. Since in about 37 percent of normal persons a venous retinal pulse is readily visible without the application of pressure, it is apparent that the diastolic venous pressure must closely approximate the intraocular tension. Obviously, further, the value of retinal capillary pressure must be somewhere between those of the diastolic arterial and diastolic venous pressures, and must be in fairly

close equilibrium with the intraocular tension.

In normal persons, Bailliart found the systolic blood pressure in the retinal arteries to be 65 to 70 mm. Hg, the diastolic pressure to be 30 to 35 mm. Hg. The venous pressure he described as about 25 mm. Hg, very close to the intraocular tension. Bailliart found that both systolic and diastolic retinal arterial blood pressures increased with age and with rising systemic blood pressure, though in the latter case the rise in retinal blood pressure was not always proportional to the rise in peripheral blood pressure. His studies of ocular disease were almost entirely limited to those phenomena associated with hypertension.

The facts presented by Bailliart as to retinal arterial blood pressure in normal and hypertensive states were promptly confirmed by a number of other observers, though there were differences in the exact measurements reported, obviously due to slight differences in the application of an unstandardized technique and to the personal equation in determining such an end point as the appearance of "maximal pulsation" in the retinal arteries.

It remained, however, for Sobanski, of Lauber's clinic in Warsaw, to adopt Bailliart's work to the problem of such conditions as choked disc, glaucoma, and optic atrophy. From the appearance of Lauber's first paper on choked disc in 1934, he and Sobanski have so far published 21 papers, of which 12 are wholly or in part devoted to a consideration of tabetic optic atrophy. These 21 papers,[61 to 80] of which 11 are by Lauber and 10 by Sobanski (although Sobanski appears to have done most of the work, Lauber reporting as Sobanski's chief of clinic), have appeared in four languages (English, German, French, and Polish) and are so repetitive that they may be considered as a group rather than singly.

Sobanski conceived the idea that various intraocular diseases might result from disturbed nutrition of the optic nerve and retina due to disturbances in the interrelationships of peripheral arterial blood pressure, retinal arterial blood pressure, retinal venous blood pressure, intraocular tension, and cerebrospinal-fluid pressure. In the normal person, Sobanski found the systolic arterial pressure in the retina ranged from 68 to 90 mm. Hg, the diastolic pressure between 40 and 56 mm. Hg (it is to be observed that these measurements are higher than those of Bailliart). Sobanski's measurements of normal venous pressure are lower than those of Bailliart (Sobanski's systolic 28 to 36 mm., diastolic 14 to 23 mm. Hg). The lowest level of arterial tension (diastolic), therefore, lies at least 14 mm. Hg above the highest normal level of intraocular tension (26 mm. Hg). This differential, say Lauber and Sobanski, is the necessary condition for normal circulation in the retina. If the differential between intraocular tension and retinal diastolic arterial pressure is diminished, there is resulting disturbance of the capillary circulation (the exact level of which is very difficult to determine, but is assumed to be above the levels of intraocular tension and venous systolic blood pressure, but below that of diastolic arterial blood pressure, therefore, according to Sobanski, between a mean of 33 and 48 mm. Hg).

In true glaucoma the intraocular tension rises and the retinal diastolic pressure remains unaltered, thereby reducing the normal differential of 14 mm. Hg; there follows, therefore, a circulatory disturbance of the optic nerve and retina. An analogous disturbance is produced by pseudoglaucoma, when the diastolic retinal arterial pressure falls while the intraocular tension remains normal. If, how-

ever, the diastolic blood pressure in the retinal arteries is high (70 mm. Hg or better), then even an increase in intraocular tension to such high sustained levels as 35 to 50 mm. Hg will not produce the circulatory change in the nerve and retina, since the differential is maintained. Glaucomatous circulatory changes in the nerve, therefore, do not develop in persons with hypertension (in whom the rise in retinal arterial blood pressure parallels the rise in systemic blood pressure). The optic atrophy in glaucoma they consider to be chiefly due to the circulatory change, and the basic difference between the atrophy of true and pseudoglaucoma lies in the excavation of the cup, which occurs only in presence of increased intraocular tension.

Though Lauber and Sobanski do not definitely say so, they apparently consider the primary optic atrophy of tabes as identical with pseudoglaucoma. In tabes, their argument is as follows: All tabetics with primary optic atrophy suffer from peripheral hypotension (diastolic if not systolic). This in turn produces retinal hypotension, and the diastolic retinal arterial pressure falls below its normal level of 40-56 mm. Hg to approach the level of intraocular tension (18-26 mm. Hg). When the differential between retinal diastolic blood pressure and intraocular tension falls much below 14 mm. Hg, circulatory failure in the optic nerve occurs, with resulting atrophy. Patients with tabes but without optic atrophy are spared this disaster because in them the peripheral blood pressure (and therefore the retinal blood pressure) is normal or elevated, and the fall in differential does not result. Hypotension is common in tabes because of the frequent association of syphilitic aortitis, and a dilated aorta usually leads to hypotension. The results of chemotherapeutic treatment of optic atrophy are usually unsatisfactory because all drugs used in the treatment of syphilis—arsenic, bismuth, mercury, the iodides—lower systemic blood pressure, as does also fever therapy; for example, induced malaria.

Assuming the validity of these arguments so far, the treatment of tabetic optic atrophy should be identical with that of glaucoma, say Lauber and Sobanski. One may approach the treatment problem in two ways, either of which is calculated to restore the normal differential of 14+ mm. Hg between diastolic retinal arterial blood pressure and intraocular tension; that is, to raise the systemic and thereby the retinal blood pressure, or to lower intraocular tension. Since the former is obviously completely impossible, no one having yet found a satisfactory method of producing sustained peripheral hypertension, the latter was adopted. The procedures advised by Lauber and Sobanski are, first, a trial of drugs calculated to lower intraocular tension, for example, pilocarpine, eserine; and if these fail, operative interference by means of cyclodialysis, which has the effect, even in the normal eye, of lowering intraocular tension to the level of 6 to 14 mm. Hg.

In support of these contentions, the documentation provided by Lauber and Sobanski is inadequate. As nearly as one can tell from a perusal of their various papers on the subject, the same patients, tabetics with optic atrophy and controls, appear in all. Sobanski claims, however, that of 33 tabetic patients with optic atrophy, the differential between diastolic retinal blood pressure and intraocular tension was 18 mm. Hg or less in 29 (but below 14 mm. Hg in only 9 instances); while in 9 tabetics without optic atrophy, studied as controls, this differential was 19 mm. Hg or more in all. No statements can be found as to normal controls, or controls with syphilis but without neuro-

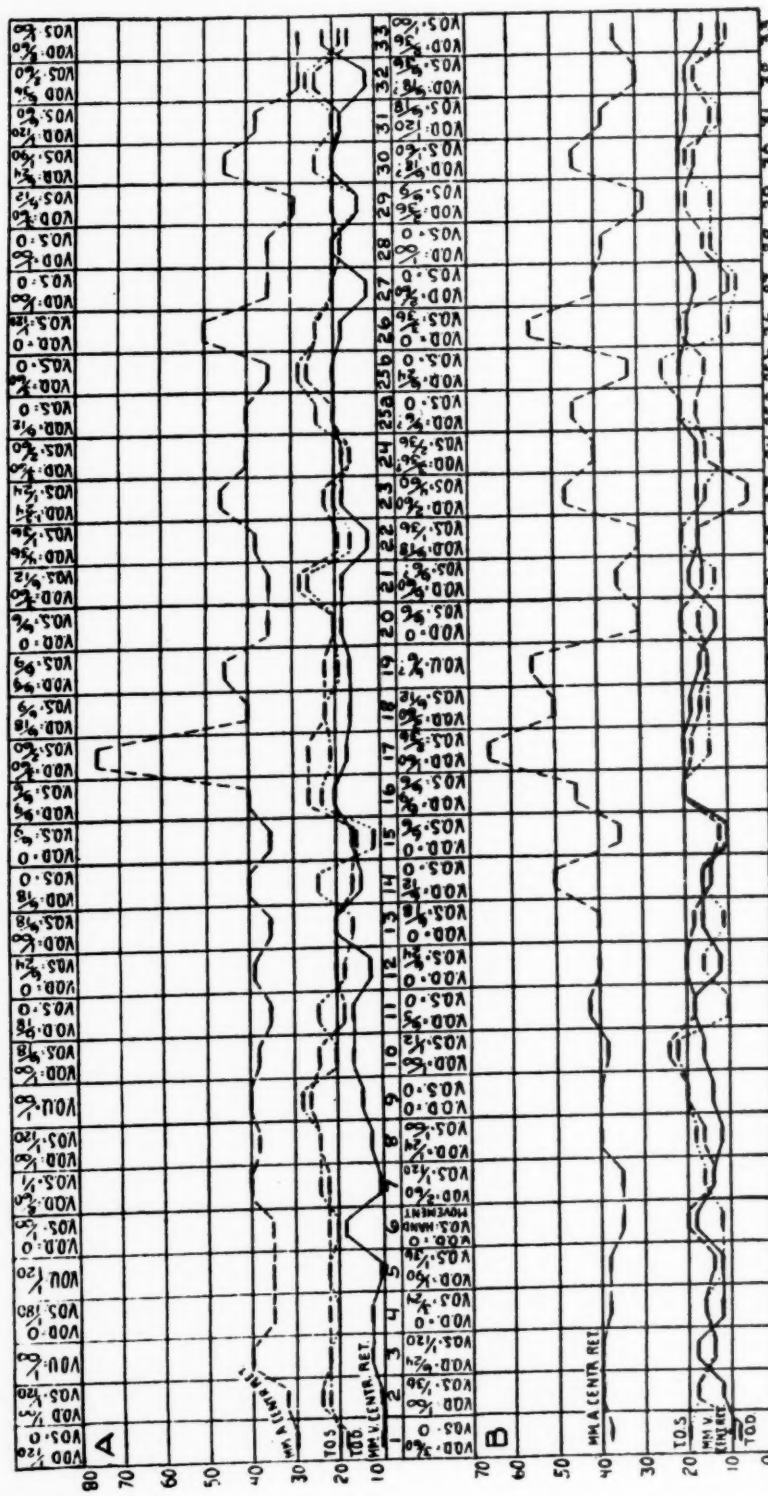


Fig. 2. Reproduced from Lauber (reference 69). Chart 5.—Data for 33 patients with atrophy of the optic nerve. *A*, condition before treatment; *B*, condition after treatment. *Mm. A. centr. ret.* indicates the diastolic arterial pressure in the retina; *Mm. V. centr. ret.*, the diastolic venous pressure in the retina; *T.O.D.*, the intraocular tension of the right eye, and *T.O.S.*, the intraocular tension of the left eye.

syphilis, or with other types of neurosyphilis than tabes.

In several papers appear identical charts dealing with the pressure findings and results of pilocarpine, eserine, and cyclodialysis in 33 cases. These charts, so frequently presented by Lauber and Sobanski themselves, are here reproduced as figures 2 and 3.

In Sobanski's latest paper (1938) these 33 cases are expanded to 55, and the results obtained from the special procedures recommended are as follows: Of the 110 eyes, 13 were blind before treatment and were not improved. Of the remaining 97 eyes, 51 improved, in 20 there was no change, while in 26 the condition became worse after a primary period of improvement. The period of posttreatment observation was less than 1 year in 37; from 1 to 2 years in 13; and from 2 to 3 years in 5 patients. No patient has so far been observed for longer than 36 months.

Most unfortunately for the importance of this theory and the practical results of treatment, and in spite of Lauber's objection that all forms of antisyphilitic treatment lower blood pressure and are therefore harmful, Sobanski says in two of his several papers (though Lauber fails to mention it in any) that all patients treated by these special procedures were also given antisyphilitic treatment, and some (number not stated) were treated with induced malaria! (*"Alle Kranken wurden antiluisch, zumeist mit Salvarsan und Wismut behandelt, vereinzelt wendeten wir Malariabehandlung an,"* Arch. f. Ophth., 1936, v. 135, p. 414). It seems obvious that under these circumstances any conclusions as to the efficacy of cyclodialysis drawn by Lauber and Sobanski are wholly unjustified.

As with any new procedure in medicine, the method was promptly repeated by other workers, whose reports fall into two general categories: those which accepted the theory and confirmed the results; and those in which the theory was subjected to a critical analysis in a more analytic fame of mind, sometimes with verification of the original results, sometimes with denial.

In the former group fall Rintelen[81] and Arruga.[82] The latter treated 15 patients with tabetic optic atrophy, of whom 9 were given pilocarpine plus medication (including atropine!) calculated to raise peripheral blood pressure and 6 were subjected to cyclodialysis. As with Sobanski's cases, all those patients with positive serologic findings in blood or cerebrospinal fluid were also given antisyphilitic treatment, and Arruga's results are therefore open to criticism and difficult to evaluate. Rintelen contents himself with acceptance of the views of Lauber and Sobanski, without documenting his own experience. Miklos[83] treated 13 patients by Lauber and Sobanski's methods, some of whom showed slight improvement in the visual fields shortly after pilocarpine administration or operation; but the observation periods in all are short; and in view of the fact that all patients received simultaneous antisyphilitic treatment, sometimes with added intraspinal injection of air and/or fever, the results cannot be evaluated. Miklos relates progressive visual failure during routine antisyphilitic treatment to such minor changes in systemic blood pressure as a drop from 130/110 to 120/90. He, like Lauber and Sobanski, appears to regard such minimal pressure changes as of pathogenetic significance, although in normal persons they occur almost hourly from such factors as meals, work, smoking, rest, or even changes in position. Albrich and Kukan[84] and Baratta,[85] were more conservative. While they did not adopt the treatment methods of Lauber and Sobanski, they did study the pressure relationships in pa-

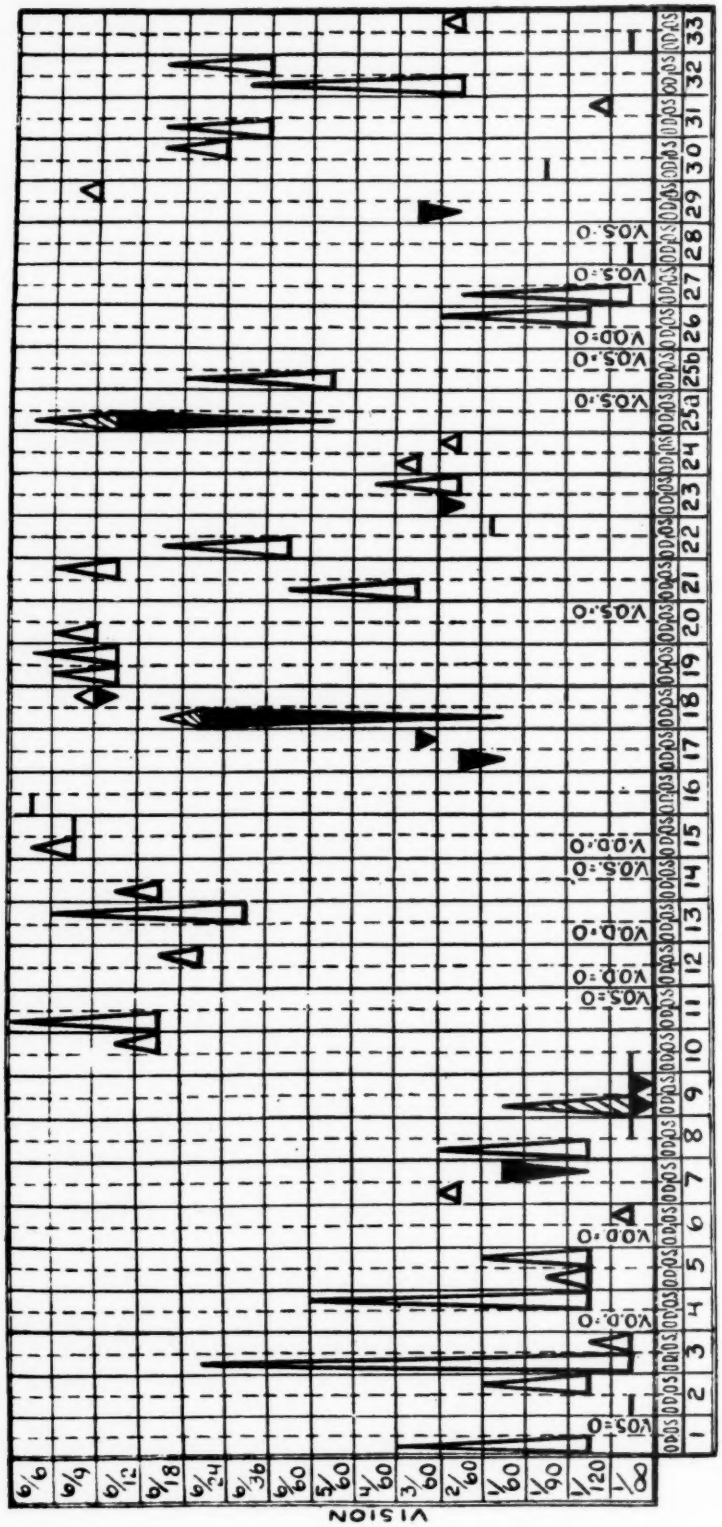


Fig. 3. Reproduced from Lauber (reference 69). Chart 6.—Changes of acuity of vision during observation of 33 patients under treatment.

tients with tabes and in controls. Albrich and Kukan point out that Sobanski's own material contradicts his theory. While Sobanski claims that if the differential between diastolic retinal arterial pressure and intraocular tension falls below 20 mm. Hg, optic atrophy will result, nevertheless in many of Sobanski's own cases the differential far exceeds this figure, and his patients thus had neither general nor retinal hypotonia. Albrich and Kukan studied the general and local blood pressure in 6 patients with tabetic optic atrophy, and in 38 persons whose eyes, except for refractive errors, were normal. In general, the values in tabetic optic atrophy were the same as those in normal persons; and in several nonsyphilitic persons with marked general hypotonia and a very slight differential between retinal diastolic arterial pressure and intraocular tension (8 to 9 mm. Hg), the eyes were completely normal.

As a result of study of the pressure relationships in 35 patients, of whom 20 had optic atrophy (only 5 syphilitic), and 15 optic neuritis or choked disc, Baratta concludes that in optic atrophy the retinal arterial pressure is increased, rather than decreased, as Lauber and Sobanski claim.

Likewise, Aumann, Brüning, and Sohr[86] were unable to confirm the Lauber-Sobanski theory in several important respects. In 80 percent of their 29 patients, the diastolic retinal arterial pressure was 42 mm. Hg or more, in contrast to Sobanski's statement that it is 42 mm. Hg or less in 80 percent. In 13 patients cyclodialysis was carried out, but the intraocular tension subsequently gradually rose to normal levels in all but 9 eyes. A few patients showed slight immediate improvement, but in a somewhat higher number there was prompt further visual failure (as to both acuity and fields).

Parts of the theory of Lauber and Sobanski are open to question on the basis of positive or negative fact. As to positive facts there are the statements that in tabetics with optic atrophy, peripheral hypotonia (diastolic if not systolic) is the rule, whereas in tabes without optic atrophy the peripheral blood pressure is either normal or elevated. This statement, offered without documentation by Lauber and Sobanski, is certainly not common medical experience and is promptly and thoroughly contradicted by Langhammerová.[87] This observer examined 153 patients with tabes, 59 of whom had optic atrophy and 94 did not. The findings as to peripheral blood pressure were as follows:

	Tabes with Optic Atrophy	Tabes without Optic Atrophy
Hypotonia	6.7 percent	24.4 percent
Normal blood pressure	69.4 percent	53.2 percent
Hypertonia	23.7 percent	22.3 percent

Thus, the incidence of peripheral hypotonia was four times greater in tabetics without optic atrophy than in those with it.

Lauber and Sobanski advance the medically curious explanation that hypotonia in tabes is due to the coexistence of syphilitic aortitis, completely overlooking the well-demonstrated fact that syphilitic aortitis does not cause hypotension or the obvious objection that if it did, and if optic atrophy depended upon this hypotension, the incidence of optic atrophy in patients with aortitis but without tabes should be high, whereas actually it is wholly lacking.

Finally Lauber and Sobanski say that all drugs used in antisyphilitic treatment lower peripheral vascular tension. This statement, also undocumented, is at complete variance with the known facts, which are that neither in normal persons nor those with hypo- or hypertension

have any of these drugs more than a transitory effect (a few minutes following injection) in any direction on the blood pressure (Moore[88]).

To explain the fact that optic atrophy does not always nor frequently occur in patients with aortic regurgitation, in whom the peripheral (and retinal) diastolic blood pressure often falls to zero, Sobanski offers the weak, and again undocumented, explanation that in these cases there is an accompanying fall in intraocular tension, which prevents the development of optic atrophy. If this fall in intraocular tension fails to occur in aortic syphilis, optic atrophy always develops, says Sobanski. Even assuming that this were true, although such a statement is most obviously open to criticism, Sobanski fails to explain why optic atrophy should not also occur in rheumatic aortic insufficiency.

Indeed if hypotonia, peripheral and retinal, were the correct explanation for the development of optic atrophy in tabes, there is no reason to believe that optic atrophy would not also occur in any condition in which prolonged hypotonia was a feature; for example, in Addison's disease, the anemias.

However, since the work of Lauber and Sobanski may have some merit, certainly in choked disc in which both retinal arterial and venous pressure appear to be an accurate index of the degree of intracranial tension, and possibly also in glaucoma, a number of observers, especially in Japan, have undertaken the sort of detailed study of the retinal circulation which, after all, forms the basis of Lauber's and Sobanski's theory. Especially interested are Suganuma and his co-workers, who present a series of papers which may well serve as a background for further and more accurate investigations of glaucoma and optic atrophy.[89 to 92] These studies, carried out on large numbers of normal people (300), have shown that when the average peripheral systolic blood pressure is 120 mm. Hg, the retinal systolic blood pressure is 59 to 65 mm. Hg, and the retinal diastolic arterial pressure 36 to 38 mm. Hg. The technique of taking retinal blood pressure can account for differences of 20 to 30 mm. Hg under identical conditions. The retinal blood pressure undergoes important changes with change in position of the subject, without concomitant change in systemic blood pressure; for example, in the recumbent position there is a 23-mm.-Hg average rise in systolic retinal blood pressure, and a 12-mm. average rise in diastolic pressure. The relationship of retinal to systemic blood pressure is a genuine one, and varies in the normal from 44:100 to 55:100. Under normal conditions the retinal blood pressure may vary markedly in the two eyes, and there is no direct relationship between normal intraocular tension and retinal blood pressure. With advancing age, the average retinal blood pressure tends to increase, as does the systemic blood pressure.

In both essential hypertension (40 cases) and the hypertension due to chronic nephritis, retinal blood pressure increases roughly proportionally to the systemic blood pressure. The highest retinal pressures observed by Suganuma were systolic 134 mm., diastolic 106 mm. Hg. According to Suganuma there is a group of cases in which retinal blood pressure is markedly increased while systemic blood pressure remains normal. This state of affairs, which he describes as isolated cerebral hypertension, has been observed in a wide variety of conditions, such as beriberi, neurasthenia, hysteria, pregnancy, epilepsy, and increased intracranial pressure.

Vancea[93] has found retinal hypotension without alterations of the systemic

blood pressure in pregnancy, tuberculosis, and after lumbar puncture.

Streiff[94] confirms the fact that, in general, the retinal blood pressure tends to follow the course of the systemic blood pressure, though his chief interest lies in the ocular effects of the systemic and retinal hypertension. He concludes that when the retinal systolic arterial pressure exceeds by 20 or more mm. Hg, the arbitrary value of one half the systemic arterial pressure, the eye usually shows some vascular lesion.

Knobloch[95] produced retinal arterial hypotonia in rabbits, and subsequently attempted to cause optic atrophy by poisoning with quinine, and by repeated bleeding, without results. On experimental grounds, he believes the Lauber-Sobanski theory to be unjustified.

Several years after the uncritical acceptance of the Lauber-Sobanski theory by a number of ophthalmologists, Ascher[96] finally provided a comparison of the findings in tabetics with and without optic atrophy, as follows:

		23 Patients with Tabetic Optic Atrophy	28 Patients with Tabes without Optic Atrophy
Intraocular tension	Average	18.6 mm. Hg	19.07 mm. Hg
	Range	13 to 23 mm. Hg	13 to 25 mm. Hg
Retinal diastolic blood pressure	Average	42.1 mm. Hg	40.2 mm. Hg
	Range	30 to 65 mm. Hg	31 to 60 mm. Hg
Systemic blood pressue	Average	133/81	131/82
	Range	110/55 to 170/110	105/55 to 200/130

Since these findings are in direct contradiction of those of Lauber and Sobanski, Ascher seems unnecessarily gentle when he suggests that the pressure abnormalities described by the Warsaw workers may not be present throughout the entire course of optic atrophy, but only during the actual period of visual failure!

Horniker[97] finds the normal diastolic retinal pressure to be 30 to 35 mm. Hg (well below the level set by Sobanski as usual in optic atrophy), and that its normal relationship to brachial diastolic pressure is 0.45 to 1. Among 67 cases of epilepsy without optic atrophy, 23 had a relative retinal hypotonia.

Serr[98] reviews the recent literature on the normal and pathologic physiology of retinal blood pressure and points out that in the study of ocular diseases, the method has many pitfalls. In normal persons, the range of retinal diastolic pressure at the hands of different observers is from 25 to 56 mm. Hg; and of systolic pressure from 50 to 100 mm. Hg. He points out that Sobanski has apparently failed to take into consideration the personal equation of the examiner, and that the conclusions of the Warsaw workers are wholly lacking in adequate documentation.

Enough has been said to show that the pathologic physiology of the retinal circulation is as yet far too imperfectly understood to justify the sweeping statements of Lauber and Sobanski, and that what is known fails to give any support or foundation to their theory.

Optochiasmal arachnoiditis

For many years, as one of us (J. E. M.) pointed out in 1932,[1] there has been a feeling on the part of neurologists and ophthalmologists alike that not all cases of primary optic atrophy in syphilitics were tabetic in origin. This opinion was naturally based on the fact that in a

considerable number of patients with optic atrophy, evidences of posterior-column damage in the cord were not only completely absent but also did not subsequently develop. By analogy from patients with other forms of neurosyphilis in whom could be demonstrated at necropsy a more or less extensive basilar meningitis, it was assumed, though without adequate pathologic proof in patients with optic atrophy, that the optic atrophy was also produced by this mechanism. Indeed, as we have already pointed out, some clinicians felt that optic atrophy presumably due to basilar meningitis had a better prognosis under antisyphilitic treatment than did the tabetic form. Some observers, also, believing that even in tabes optic atrophy depended on basilar meningitis, held this belief so firmly that when necropsy failed to demonstrate such a lesion, it was assumed to have been previously present but to have healed!

Until the last decade, the relationship of basilar meningitis to optic atrophy has rested in the realm of hypothesis. Since 1929, a considerable literature has developed on the subject of adhesive optochiasmatic arachnoiditis, some cases of which are apparently due to syphilis.

Adhesive arachnoiditis involving various parts of the nervous system has been well known for many years. The form affecting the spinal cord is well described by Störring,[99] Alajouanine, Hornet, and André,[100] and most recently and best accessible to American readers by Mackay.[101] Syphilis is only one of the causes of this condition, other infections and trauma being commoner etiologic factors.

Arachnoiditis involving the optic chiasm was apparently first described in 1924 by Horrax,[102] then of Cushing's clinic, who described 33 cases of cisternal arachnoiditis simulating cerebellar tumors. The significance of this report for the development of optic atrophy was, however, not appreciated until 1929, when, almost simultaneously, Cushing and Eisenhardt,[103] Holmes,[104] and Balado and Satanowsky[105] all described optic atrophy due to arachnoiditis about the chiasm. To Balado apparently belongs priority, since he was first to operate on such a patient with the idea of releasing the adhesions about the optic nerves.

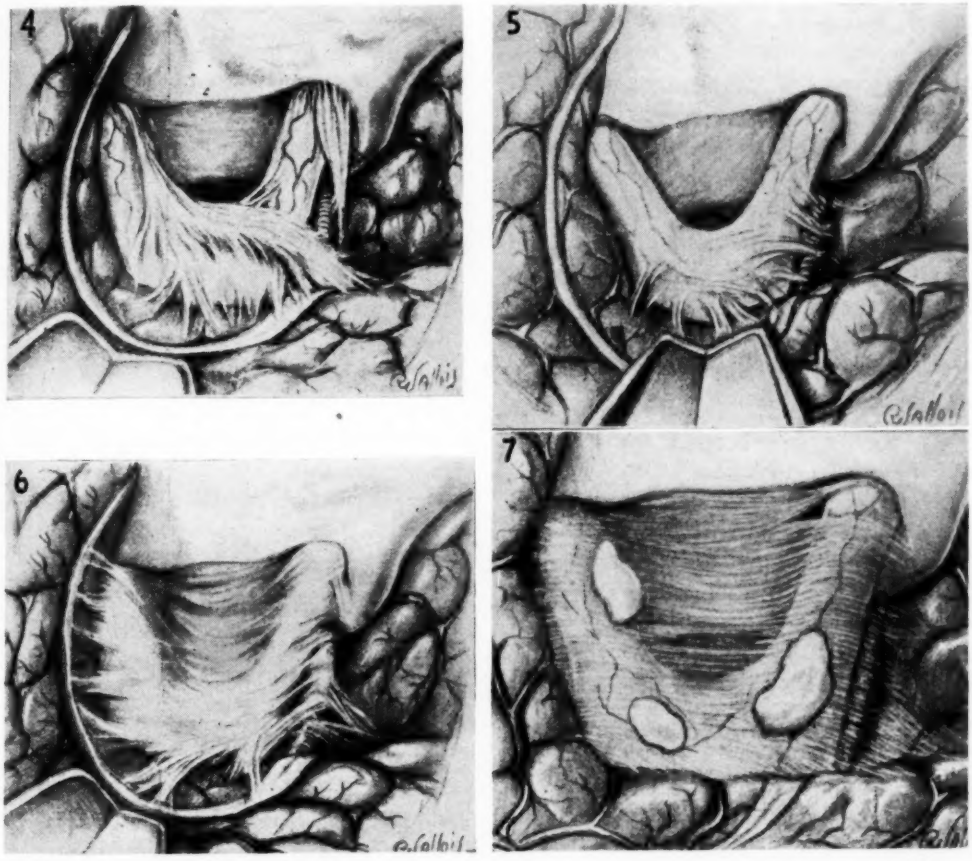
The literature on the subject rapidly grew, so that in 1937, Bollack, David, and Puech[106] were able to publish a monograph with a bibliography of 152 references, reporting 66 personal cases and 63 gathered from the literature. The discussion that follows is based largely on their work, and on a number of papers that have since appeared.

Optochiasmatic arachnoiditis produces optic atrophy by the production of inflammatory tissue—an arachnoid "veiling"—which surrounds, compresses, and fixes to neighboring structures the optic nerves and chiasm. In the mildest cases, there are very fine fibers of arachnoid springing from the vessels to, over, and around the nerves. In more advanced cases, there may be solid bands or veritable blankets of arachnoid which completely envelop the chiasm and nerves. Under these adhesions, there may or may not be serous meningitis, sometimes with the production of arachnoid pockets or cysts. Bollack and his associates published several drawings (semidiagrammatic) of the findings at operation, some of which are reproduced here as figures 4 to 7.

The etiology of optochiasmatic arachnoiditis is agreed by all observers to be extremely diverse. Trauma, encephalitis, sinus infection (ethmoid and sphenoid), other acute and chronic infections (whether intra- or extrameningeal), and syphilis are all stated to be causative agents.

In about 20 percent of the cases (15 of

the 129 reported by Bollack, David, and Puech), syphilis is a possible etiologic cause. It is a striking fact that in the cases personally reported or assembled by Bollack and his associates, however, and in most of the cases subsequently reported (excluding the papers by Haustion and information as to lesions of syphilis elsewhere in the nervous system than the optic nerve, or elsewhere in the body, being completely lacking. So far as the role played by arachnoiditis in syphilitic optic atrophy is concerned, the field is still wide open for study. It is, how-



Figs. 4 to 7. The operative findings in several cases of optochiasmal arachnoiditis (reproduced from Bollack, David, and Puech, reference 106).

mann and Vail to be referred to presently), data concerning syphilis are very sketchy indeed. In many patients, no investigation of any sort for syphilis has apparently been made; and in the majority of those diagnosed as syphilitic, the diagnosis hinges on the presence of a positive serologic test for syphilis of the blood only, cerebrospinal-fluid examinaever, generally agreed that in most such patients there is at least no obvious evidence of syphilis. One gets the impression, rarely clearly stated in the extensive literature, that optochiasmatic arachnoiditis may be a reasonably frequent cause of optic atrophy in that large group of patients so far diagnosed as "due to unknown causes" (that is, not due to tabes,

brain tumor, multiple sclerosis, or toxic amblyopia).

As to tabes, Bollack, David, and Puech say that the primary optic atrophy of this condition is usually not due to adhesive arachnoiditis, though in a small number of cases the two conditions may coincide. In this case, they point out, it is, of course, possible that a banal arachnoiditis may be superimposed on neurosyphilis.

The symptoms of optochiasmal arachnoiditis are primarily ocular. Visual failure is the primary and often the sole manifestation. It is often abrupt in onset, usually bilateral from the start, and rapidly progressive. Complete blindness may develop in a few weeks or months. The course is sometimes variable with periods of alternate aggravation and improvement. Other symptoms are rare, though headache (habitually bifrontal), pain behind the eyes, somnolence, nausea and vomiting, vertigo, polydipsia, polyuria, and obesity may infrequently occur.

The physical signs are also almost entirely ocular. Ophthalmoscopic examination may reveal simple atrophy of the optic discs (38 percent) sometimes with hazing of the disc outlines (16 percent), papillomacular-bundle atrophy (7 percent), papillary stasis (10 percent), simple hyperemia (7 percent), partial horizontal atrophy (4 percent), normal discs (10 percent), discs difficult to interpret (8 percent).* Vail,[107] in a more recent paper, insists that an "appearance of atrophy of the disc which lies between the primary and secondary type of atrophy is highly suggestive of arachnoiditis involving the chiasm."

As to other ocular manifestations, pupillary changes (except in the cases due to syphilis) are conspicuous by their absence. Likewise rare are paralyses of the oculomotor or superior branch of the fifth nerves.

The visual fields are most important and usually show changes indicative of chiasmal involvement. Unlike the course of events in hypophyseal tumor, however, the modifications in the fields may be more or less incomplete, irregular, and variable. In the cases studied by Bollack and his associates, the following alterations were found: hemianopsia 29 percent (bitemporal 17 percent, binasal, 7 percent, homonymous 5 percent), concentric constriction 23 percent, central scotoma 31 percent, horizontal constriction 5 percent, fields normal, irregular, unobtainable, or not described 12 percent. The central scotomas are almost always absolute and bilateral.

One of the outstanding features of optochiasmatic arachnoiditis is the essentially negative examination of the nervous system. The blood serologic test for syphilis and the cerebrospinal fluid are usually normal, though there may be a slight increase in cells and protein.

Roentgenologic examination shows normal skull, sella, and optic canals. Ethmoid and sphenoid disease is, however, often demonstrable. Ventriculography with air is usually normal; when iodized oil is introduced into the ventricular system, there may be complete or partial disappearance of the infundibulum, or the oil in this area may be broken up into small particles (Carillo[108]).

The points aiding in differentiation between the optic atrophy produced by arachnoiditis at the chiasm and by tabes dorsalis are briefly summarized in table 1.

Other conditions requiring differentiation from optochiasmatic arachnoiditis are tumor, disseminated sclerosis, other forms of retrobulbar neuritis, neuromyelitis optica, and Leber's disease. In many instances, say Bollack and his co-work-

* The percentage figures are taken from Bollack, David, and Puech.

ers, the situation is so confusing that the diagnosis can be made correctly only at operation.

The only treatment of the condition is surgical, with release of the adhesions. The possibility of improvement depends on the age of the lesion, visual acuity at the time of operation, and the degree of visible optic atrophy present. In an occasional case, improvement has occurred even when the optic atrophy was apparently complete and the patient already blind. Forty-eight of the 129 patients reported by Bollack, David, and Puech improved after operation. Among the patients subjected to operation were 15 in whom the lesion may have been syphilitic. They will be subsequently considered, together with patients more recently reported by other workers.

Since the publication of the monograph of Bollack, David, and Puech a number of other contributions on optochiasmatic arachnoiditis have appeared. These, together with certain references omitted by Bollack and his associates, will be briefly considered.

Morea (1933),[109] in a general paper on the subject, thinks that the lesion is often due to syphilis. He describes 10 patients (see table 2), only one of whom, however, apparently had syphilis. Malbrán and Balado (1933)[110] describe a non-syphilitic case.

Drake (1934)[111] reported a patient with tabes dorsalis, primary optic atrophy, and binasal hemianopsia. Contrary to the frequency of this form of hemianopsia in optochiasmatic arachnoiditis, it is excessively rare in tabetic atrophy, only six cases having been previously reported. Drake commented on the fact that in only one of the seven reported cases, however, did operation or necropsy

TABLE 1

DIFFERENTIATION BETWEEN THE OPTIC ATROPHY OF THE OPTOCHIASMATIC ARACHNOIDITIS AND TABES

	Optochiasmatic Arachnoiditis	Tabes
Onset	Often abrupt.	Usually insidious.
	Usually bilateral.	Often unilateral.
Course and evolution	May remain unilateral.	Always becomes bilateral.
	Capricious—alternate aggravation and improvement.	Steady progressive visual failure.
	Rapid—blindness in weeks or months.	Relatively slow—blindness in years.
	May spontaneously arrest short of complete blindness.	Complete blindness inevitable if untreated.
Visual fields	Hemianopsia (bitemporal, binasal, or homonymous) common (29 percent).	Hemianopsia very rare.
	Concentric constriction (23 percent).	Also common.
	Central scotoma (31 percent).	Also common.
Optic disc	Usually simple optic atrophy but often with hazing of borders and constriction of vessels.	Simple optic atrophy without hazing or constriction.
	Visual failure often out of proportion to disc pallor.	Pallor often more marked than visual failure suggests.
Pupils	Usually normal.	Almost always abnormal.
Other cranial nerves	Usually normal.	Often involvement of Nerve III, V, and VI.
Nervous system	Usually normal.	Usually spinal-cord damage.
Blood STS and CSF	Usually normal.	Usually positive for syphilis.
X-ray skull	Negative.	Negative.
X-ray sinuses	Ethmoids and sphenoids often cloudy.	Usually negative.
Ventriculography (air)	Usually negative.	Usually negative.
Iodoventriculography	May show obstruction to third ventricle.	No data.

rule out tumor (and presumably also arachnoiditis).

Lo Cascio (1934)[112] reported a presumed, unverified case of arachnoiditis in a nonsyphilitic patient; and a similar nonverified case was described by Platania (1936),[113] together with a review of 58 cases from the literature (covered more fully by Bollack *et al.*). Rubino (1937)[114] reported five cases, all nonsyphilitic, two of which were verified at operation.

Balado and Franke (1937)[115] described the pathologic findings at necropsy in a nonsyphilitic patient operated upon by Malbrán. There was chronic inflammation with round-cell infiltration of the arachnoid, extending into the interfascicular septa of the optic nerve, together with demyelination and disappearance of the axis cylinders in the nerve.

In 1937 there was an outpouring of papers from Vincent[116] and his associates, Desvignes,[117] Puistienne de Gandt,[118] and Thieffry,[119] all of which are devoted to repetitive general considerations with no new data. However, Vincent, Jeandelize, and Bretagne (1937)[120] report a single interesting case. A patient with tabes dorsalis and primary optic atrophy, in whom visual failure had progressed in spite of chemotherapeutic and malaria treatment to 3/100 right and light perception left, was operated upon. In spite of the fact that the general neurologic picture was clearly tabes, there was a marked arachnoiditis about the optic nerves and chiasm. The freed nerves were white and thick. The authors believe, on the basis of this experience, that when visual failure in tabes is progressive in spite of all treatment efforts, operation should be tried.

In three papers devoted to the general subject of optochiasmatic arachnoiditis, Fasiani and Belloni (1936-1937)[121 to 123] repeat the details of two patients with tabes operated upon, with extensive chiasmal arachnoiditis discovered, but without improvement as to vision.

Thiebaut and Delaitre[124] report operations on two patients with neurosyphilis (one with supposed syphilitic meningitis, the other with the type of neurosyphilis undefined) and optic atrophy, both of whom showed adhesive arachnoiditis, but the results cannot be evaluated because of the brief observation period.

François[125] reported the case of a 29-year-old man who, one-and-a-half years after infection with syphilis, developed choked discs followed by secondary atrophy. Operation showed chiasmal arachnoiditis but did not improve vision.

The most important and significant papers on the possible relationship of optochiasmal arachnoiditis to syphilis have come from American workers. Hausman (1937)[126] says "In syphilis of the central nervous system, meningitis at the base of the brain is a frequent occurrence, especially around the chiasm and in the interpeduncular region. . . . When the optic nerves and the chiasm alone are involved, the arachnoid nature of the syndrome of atrophy of the optic nerve and visual fields defects with no palsy of the other cranial nerves is frequently unsuspected and mistaken for parenchymatous syphilis; that is, tabes dorsalis. In such cases of syphilis, when all other chiasmal lesions have been ruled out, the underlying process is arachnoiditis, and the imminent danger of blindness may be arrested by surgical intervention." He reports five cases, one verified by operation (table 3) and one at autopsy, the others unverified.

Vail has published two papers on the subject. In the first,[127] he insists upon the importance of a mixed type of atrophy in the diagnosis of optochiasmal arachnoiditis. By this he means "the outline of the disc is sharply defined, the

lamina cribrosa is visible, but the caliber of the vessels is markedly reduced"; or "a blurred margin of the disc, normal sized vessels, and the presence or absence of the lamina cribrosa associated with either complete or sector atrophy of the nerve head." He feels that the diagnosis of syphilitic optochiasmatic arachnoiditis may be safely made when this type of optic atrophy is accompanied by (a) rapid loss of vision, (b) chiasmatic field defects, and (c) history or positive serologic findings of syphilis.

In his second paper,[128] Vail reports seven cases in which he has made this diagnosis. Only one (table 3) has been verified at operation. The 12 cases reported by Hausman and Vail provide far more information than most of those in the French, Italian, or South American literature, and are therefore analyzed in detail in table 2. A study of this table reveals that even in these cases there are some inadequacies of study. In a few there are definitely inadequate descriptions of the general neurologic picture. The spinal fluid was not examined in three of Vail's patients. In 7 of the 12 patients of the combined series there was no X-ray examination of the skull, and in only one was ventriculography performed. Where antisyphilitic treatment was given, no sufficient data are provided by either author as to the type and amount. A critical observer would certainly hesitate at the diagnosis of optochiasmatic arachnoiditis in Hausman's case 3, and would object entirely to the diagnosis of syphilis on the basis of the information provided in his case 4. In only 3 cases of the 12 was the diagnosis verified by operation or necropsy (Hausman's cases 1 and 5, Vail's case 1). At this stage of our knowledge of chiasmal arachnoiditis in syphilis, it seems unwise to present unverified case reports as positive diagnoses.

However, assuming the clinical impressions of optochiasmal arachnoiditis to be correct in all 12 cases, one finds interesting correlations with the general picture of neurosyphilis. One patient definitely (Vail's case 2) and one probably (Hausman's case 2) had tabes dorsalis. Six had various symptoms or signs justifying the classification of late diffuse meningovascular neurosyphilis. Three had completely normal neurologic examinations except for the ocular findings; and in one, no data are provided.

In one of Hausman's patients the fundi were normal (this, case 3, is a very dubious case). The remainder of Hausman's cases showed simple primary optic atrophy; all of Vail's cases showed what he describes as a mixed type of atrophy.

The visual fields in Vail's cases showed in one (case 1) early concentric constriction with a possible left relative central scotoma; in one (case 2) homonymous hemianopsia; and in one (case 7) a horizontal field defect. In the remaining cases (3, 4, 5, and 6), one eye was already blind or nearly so at the time of the examination; but the character of the defect in the remaining eye was such as to suggest that if earlier study had been possible, either binasal or bitemporal hemianopsia would have been found.

On the whole, these patients, as reported by Vail and Hausman, are relatively unconvincing. One wishes for more instances of operative or post-mortem verification. In all of Vail's cases, operation is subsequently planned if antisyphilitic treatment does not arrest the process.

All of the 23 patients so far operated upon for so-called syphilitic optochiasmal arachnoiditis are summarized in table 3. Seven were blind, or nearly so, at the time of operation. In all, the neurosurgeon reported the finding of chiasmal arachnoiditis. The operation was a total

TABLE 2

AN ANALYSIS OF THE CASES OF OPTOCHIASMAL ARACHNOIDITIS REPORTED BY HAUSMAN (REFERENCE 126) AND VAIL (REFERENCE 128)

Author	Race	Sex	Age	Evidence of Neurologic Disease other than Optic Atrophy	Duration Visual Failure on Adm.	Fundi	Visual Acuity		Fields	Blood STS	Cerebrospinal fluid			
							O.D.	O.S.			Cells	Prot. or Glob.	Wa.R.	Coll. curve
Hausman #1	C	F	26	Headache, anemia, pupils sluggish, neurologic examination otherwise negative	2 yrs.	Bilateral primary O.A.	20/100	L.P.	Bitemporal hemianopsia	4	20	+	0	0
Hausman #2	W	M	40	Loss libido & potentia; girdle pains. Pupils inactive to light. Neurologic examination otherwise negative	2 yrs.	Bilateral primary O.A.	2/100	0	Only small sector retained in lower nasal quadrant, right.	4	4	+	0	0
Hausman #3	W	M	41	Convulsions 18 yrs.; possible diabetes insipidus. Memory loss & insomnia; deep reflexes exagerated. Babinski bilaterally +	0	Normal	N.D. presumably normal	N.D.	Tendency to temporal hemianopsia left. Concentric constriction both	4	4	±	0	0
Hausman #4	W	M	31	Trigeminal neuralgia 6 years; otherwise negative	2 yrs.	Bilateral primary O.A.	2/10	2/10	Binasal hemianopsia	0	0	40 mg.	0	0
Hausman #5	W	F	28	Unilateral headache 11 yrs., tinnitus, vomiting; otherwise neurologic examination negative	6 wks.	Bilateral papilledema	N.D.	N.D.	Concentric constriction, with tendency to nasal hemianopsia, right	4	N.D.	N.D.	2	Mid-zone
Vail #1	W	M	58	Pupils dilated, inactive to light	1 yr.	Mixed-type atrophy	0	15/15	Possible relative central scotoma O.S.	4	N.D.	N.D.	N.D.	N.D.
Vail #2	W	M	35	Tabes dorsalis	1 mo.	Mixed-type atrophy	20/15 −1	F.M. 6 ft.	Homonymous hemianopsia	4	90	+	4	Mid-zone
Vail #3	W	M	61	No data	2–3 yrs.	Mixed-type atrophy	20/30	H.M. 2 ft.	Upper temporal sector defect	4	No data			
Vail #4	C	F	27	Old right hemiplegia; left N III and right N VII palsies; pupils inactive	N.D. "Eyes always poor"	Mixed-type atrophy	20/70	0	Temporal defect	3	N.D.	165 mg./%	3	+ No details
Vail #5	W	M	64	Old left hemiplegia	1 mo.	Mixed-type atrophy	5/200	20/100	Nasal defect	4	No data			N.D.
Vail #6	C	M	42	Normal (incomplete statement). Hypertension 220/140	2 mos.	Mixed-type atrophy	L.P.	20/30	Possible nasal defect	4	No data		4	+ No details
Vail #7	C	M	55	Neurologic examination negative. Right pupil sluggish	1 yr.	Mixed-type atrophy	4/200	20/100	Horizontal defect, lower fields largely destroyed	4	130	99 mg./%	4	+ No details

X ray	Ventriculography	Antisyphilitic Treatment Duration and Amount	Result as to Vision	Operative Findings	Final Visual Acuity O.D.	Final Visual Acuity O.S.	Final Fields	Duration Postoperative Observation	Postoperative Complications	Remarks
Osteitis of right parietal bone, sella large	N.D.	0		Chiasm matted down with adhesions; these were freed	20/100	F.M. −1	Expanded	5 mos.	Diabetes insipidus; Jacksonian fits mouth	
Enlargement of sella	Low-grade internal and external hydrocephalus	Neo and Hg, 3 mos.	Steadily worse; practically blind							
Normal	N.D.	"Intensive"; type and amount not stated	4 yrs. later, fundi normal but V.O.D. 20/40−1, V.O.S. 20/50 +1, fields slightly worse							Highly dubious case.
Normal	N.D.	8×Neo, 15×Hg	Slight improvement in fields							First diagnosed as O. A. 6 yrs. before symptoms of visual failure, when V.O.D. 10/40, V.O.S. 10/100. Serologic tests blood and C.S.F. always negative. Diagnosis of syphilis highly dubious
N.D.	N.D.	Rx. given, type and amount not stated	1 yr. later, papilledema 4 diopters persists. Fields worse. V.O.D. 20/60, V.O.S. 20/50	6 mos. later, semistuporous. Right parietal decompression; death 12 hrs. later						Necropsy showed gumma right parietal lobe with perichiasmal syphilitic plastic meningitis
N.D.	N.D.	Rx. given, type and amount not stated; also malaria	5 yrs. later, V.O.D. 0, V.O.S. fingers at 8 feet	Thin membrane not unlike filter paper covering chiasm and blood vessels	0	F.M. at 10 feet	Improved	2 mos.		
N.D.	N.D.	Rx. given, type and amount not stated; also malaria	7½ mos. later, progressive visual failure							
N.D.	N.D.	Rx. given, type and amount not stated	2 mos. later, visual fields slightly improved, but no further data							
N.D.	N.D.	No data								
N.D.	N.D.	N.D.								
N.D.	N.D.	N.D.								
Negative	N.D.	N.D.								

TABLE 3

PATIENTS WITH SYPHILIS OPERATED UPON FOR OPTOCHIASMATIC ARACHNOIDITIS

Case No.	Bollock, David, and Puech's Case #	Author Reporting	Duration Optic Atrophy	Visual Acuity Before Operation		Fields	Visual Acuity After Operation		Summary of Result of Operation	Period of Observation
				O.D.	O.S.		O.D.	O.S.		
1	1	Cushing	3 yrs.	2/5	2/200	Concentric constriction; scotomas	Unchanged		Failure	N.D.
2	38	Bailliart, David, Schiff-Wertheimer	7 mos.	0	0	—	1/50	0	Improved	1 mo.
3	39	Spaeth	N.D.	6/5	6/15	Binasal hemianopsia, scotomas	N.D.	N.D.	Temporary increase in fields. Doubtful	6 mos.
4	47	Vincent and Hartman	N.D.	F.M.	5/15	Binasal hemianopsia	Unchanged		Stationary	3 mos.
5	9a	Bollack, David, and Puech	5 mos.	5/7	5/5	Concentric constriction	5/5	5/5	Fields normal. Improved	2 mos.
6	11a	Bollack, David, and Puech	22 mos.	1/200	1/200	—	Blind		Failure	4 mos.
7	19a	Bollack, David, and Puech	N.D.	1/35	1/100	Bitemporal hemianopsia	1/25	1/100	Improved	3 wks.
8	23a	Bollack, David, and Puech	10 mos.	0	1/100	Binasal hemianopsia	0	L.P.	Failure	N.D.
9	27a	Bollack, David, and Puech	2 yrs.	0	H.M.	—	Blind		Failure	N.D.
10	30a	Bollack, David, and Puech	1 mo.	1/10	4/10	N.D.	"No improvement"		Failure	N.D.
11	38a	Bollack, David, and Puech	15 mos.	0	5/25	Concentric constriction; scotomas	Died at operation		Death	—
12	49a	Bollack, David, and Puech	23 mos.	F.M.	0	Concentric constriction	0	0	Failure	6 mos.
13	53a	Bollack, David, and Puech	18 mos.	5/50	5/25	Central scotomas	5/50	5/50	Arrest	3 wks.
14	54a	Bollack, David, and Puech	7 mos.	0	1/20	Binasal hemianopsia	0	F.M.	Failure	3 wks.
15	62a	Bollack, David, and Puech	9 mos.	2/50	H.M.	Bitemporal hemianopsia	2/50	1/100	Improved	3 mos.
16		Morea	2 yrs.	1/2	1/1	Concentric constriction	No operative follow-up		N.D.	—
17		Vincent, Jeandelize, and Bretagne	N.D.	3/100	L.P.	N.D.	Unchanged		Failure	N.D.
18		Fasiani and	Details unavailable			No details	No details		Failure	N.D.
19		Belloni	Details unavailable			—	No details		Failure	N.D.
20		Thiebaut and	5 mos.	6/6	1/7	Bitemporal hemianopsia	Unchanged Fields sl. better		Improved	3 wks.
21		Delaitre	4 mos.	1/10	1/10	Bitemporal hemianopsia	N.D.	N.D.	N.D.	6 days
22		Hausman	2 yrs.	20/100	L.P.	Bitemporal hemianopsia	20/100–1 Fields expanded	F.M.	Improved	5 mos.
23		Vail	5 yrs.	0	F.M.	Central scotoma O.S.	0 Fields improved	F.M.	Improved	2 mos.

Abbreviations: N.D.=No data; F.M.=Finger movements; H.M.=Hand movements; L.P.=Light perception.

failure in 10 cases. One patient died postoperatively. No data as to outcome are available in two patients. In 10, vision is said to be either stationary or improved as a result of operation. It is to be noted, however, that in no case are the postoperative observation periods long enough to justify such statements. Before one can be even partly sure of arrest or improvement in syphilitic primary optic

atrophy, a minimum observation period of one year, preferably three years, must elapse. In these cases from the literature, the observation period of supposed favorable results ranged from a minimum of three weeks (three such cases!) to a maximum of five months (Hausman's case 1).* Obviously, these time periods are far too short to justify any statements as to the probable outcome.

In reviewing the entire literature relating to optochiasmatic arachnoiditis as it relates to syphilis, one gets the impression that practically all reported cases have been inadequately studied either from the neurologic, syphilogic, radiologic, or serologic standpoints, or at times from all of these; that too many cases are reported on presumption and without verification; and that the results of operation are, to say the least, not very hopeful. Nevertheless, the field is a fruitful one for further study; and it is to be hoped that neurologists, syphilologists, and ophthalmologists the world over, working in close coöperation with neurosurgeons, will arrange for operation in those patients with primary optic atrophy, with or without chiasmal field defects, in whom progressive visual failure occurs in spite of *modern*[21] antisyphilitic measures (that is, malaria and/or subdural treatment). Probably especially suitable for such operative interference are those neurosyphilitic patients in whom optic atrophy appears as an isolated neurologic phenomenon, without evidence of posterior-column damage to the spinal cord; although a series of frank tabetics should also be studied.

* In Hausman's paper, the reported observation period after operation was only 13 days, but in a personal communication, he states that the patient has held the reported gain for five months.

The relationship of optic atrophy to nutritional deficiency

The new theories so far discussed; that is, vascular spasm (Abadie), disturbance of vascular and intraocular pressure relationships (Lauber and Sobanski), and optochiasmal arachnoiditis, may (especially the latter) suffice to explain the etiology and suggest the rationale of treatment of a few cases of syphilitic primary optic atrophy. None of these theories, however (including that of Lauber and Sobanski which purports to do so), is adequate to explain the one outstanding fact, that in neurosyphilis, primary optic atrophy is associated in the overwhelming majority of cases with a single form of the disease, tabes dorsalis. It seems reasonable to assume, therefore, that the etiologic background of tabetic optic atrophy is identical with that of tabes itself.

It is necessary to repeat that tabes differs from all other forms of neurosyphilis in at least five important respects: (1) Its longer incubation period. (2) Its extraordinary selectivity for certain tracts of brain and spinal cord. (3) Its relative refractoriness to treatment. (4) Its tendency toward absolute or relative seronegativity. (5) The relative paucity or complete absence of *T. pallidum* from the diseased areas in the nervous system.

Efforts to explain the etiologic background of tabes have so far centered largely on the toxin theory of Hauptmann,[129] which, however, rests on pure hypothesis with no shadow of clinical or experimental proof.

At this point, it is important to note that tabes has not been overlooked by the anthropological students of the relationship of disease to human constitution. The old familiar clinical observation that

tabes tends to occur in persons of asthenic habitus, while paresis, on the contrary is more frequent in those of pyknic or sthenic habitus, has received statistical confirmation from the careful work of such observers as Curtius, Schlotter, and Scholtz.[130] Tabetics are habitually ill-nourished persons. From the study of tabetic patients themselves, it has not yet been made clear, however, whether tabes *develops* primarily in persons of asthenic habitus, or whether the asthenic habitus is an *aftermath* of tabes.

In at least one respect, the selectivity of the lesions in the nervous system, tabes bears a close clinical analogy to certain neurologic diseases now known certainly to be due to nutritional deficiency; for example, the subacute combined sclerosis of pernicious anemia, the peripheral neuritides of certain vitamin-B deficiencies. Indeed, in some cases diagnostic differentiation between tabes and subacute combined sclerosis is often extraordinarily difficult. To pursue the clinical analogy between tabes and the various deficiency peripheral neuritides is difficult because of lack of adequate pathologic information concerning the peripheral nerves in tabes. Certain tabetic symptoms, especially paraesthesias and lightning pains, suggest peripheral-nerve involvement; but modern pathologic study is essential to determine the point.

The only forms of nutritional deficiency known definitely to produce neurologic lesions are those due to vitamins A and B. Recent reviews of the relationships of the latter are supplied by Lewy[131] and by Williams and Spies.[132] Familiar neurologic changes in diseases certainly or possibly associated with vitamin-B deficiency are: (1) Peripheral neuritis (beri-beri, alcoholism, pregnancy); (2) posterior-column or combined-tract degeneration in the spinal cord, with or without degeneration in the anterior and posterior horns (pellagra, pernicious anemia, diabetes); (3) retrobulbar neuritis (beri-beri, alcoholism, lead poisoning, pregnancy, pellagra, diabetes) sometimes followed by optic atrophy.

In none of the supposed deficiency diseases does primary optic atrophy occur so typically or so frequently as in tabes dorsalis. As to pernicious anemia, there are singularly few reports of optic atrophy. Cohen[133] reports two cases of primary anemia with optic atrophy, in one of which the relationship seemed definite, in the other probable. Both patients improved on treatment with liver. Kampmeier and Jones[134] describe 3 cases of primary optic atrophy among 233 patients with pernicious anemia, 74 percent of whom had some other evidence of neurologic damage. In their large clinical material, the incidence of optic atrophy was relatively very infrequent, though they quote W. R. Brain's textbook statement (unverified by other clinicians) that optic atrophy occurs in about 5 percent of all patients with pernicious anemia. Certainly, general experience indicates that in primary anemia optic atrophy is neither so frequent nor so rapidly and certainly progressive to blindness as in tabes.

Courville and Nielsen[135] add two more instances of optic atrophy associated with subacute combined degeneration of the cord.

Moore has published a series of papers[136] describing a syndrome observed among school children and poor natives of Nigeria, consisting of sore tongue and mouth, scrotal or vulvar scaling and itching, and severe retrobulbar neuritis progressing to optic atrophy. In most instances there is serious impairment of vision with the ophthalmoscopic picture of papillomacular bundle or later complete primary optic atrophy; but visual failure usually stops short of complete

blindness. He believes that this condition is associated with dietary deficiency, probably though not definitely vitamin B, since the retrobulbar neuritis may be ameliorated or cured by the administration of "marmite" or fresh or autoclaved yeast. Certainly it is not due to vitamin-A deficiency, he says, since xerophthalmia is never associated, and since the usual native diet contains large quantities of palm oil rich in vitamin A. The condition is apparently extraordinarily frequent in Nigeria, since Moore says he has seen "thousands of cases"; and it probably occurs in other tropical areas as well. Moore says identical or analagous retrobulbar neuritis has been reported from the West Indies (Clark, Scott, St. John), the Solomon Islands (Meagher), Malaya (Landar and Pallister), Gold Coast (Purcell), Sierra Leone (Wright), and Ceylon (Nicholls).[137]

Much more stimulating than the clinical reports of optic atrophy in deficiency diseases is the experimental work that has been done in this field. In 1929, Hughes, Lienhardt, and Aubel[138] fed young pigs from the time of weaning on white corn, tankage, and bone ash (a diet deficient in vitamin A). In 6 to 10 months, these animals became blind and developed incoördination and spasms. At necropsy, degenerative changes were found in the spinal cord, and in the optic, sciatic, and femoral nerves. Since the addition to the diet of cod-liver oil, butter fat, yellow corn, and alfalfa-leaf meal prevented or relieved the condition, the preventive factor was thought to be vitamin A. This is the first report that can be found of optic atrophy produced by deficient diets.

Mellanby was next to work in this field. In 1931, he reported[139] experiments with young puppies. To these animals he fed diets containing large amounts of cereal other than yellow maize, and deficient in vitamin A or carotene. After variable times, there developed spasticity, ataxia, and weakness of the extremities. The addition to the deficient diet of 2-5 gm. of ergot daily hastened and intensified the clinical and pathologic picture. The presence in the diet of any source of vitamin A, such as liver-oil, whole milk, butter, egg yolk, or some source of carotene, such as green vegetables, carrots, or carotene itself prevented or diminished the degenerative changes even though ergot was eaten. At necropsy there were scattered degenerative changes in the spinal cord (not limited to the posterior columns) in the form of demyelinization of the nerve fibers. Mellanby felt that the variability of onset of neurologic symptoms was due to the large but variable reserve of vitamin A in the liver, and that damage did not result until this reserve was dispersed.

In discussing the possible relationship of these experiments to clinical medicine, Mellanby mentions the neurologic manifestations of convulsive ergotism, lathyrism, pellagra, disseminated sclerosis, pernicious anemia, and tabes dorsalis. His statement as to tabes is short but important, since, so far as is known, it is the first suggestion in the literature that nutritional deficiency may play an etiologic role in this disease. He says "There are the degenerative cord changes found in tabes dorsalis and general paralysis and said by these same workers (Orr and Rows) to arise from toxins ascending the peripheral nerves in the lymph stream which must receive consideration from this new angle."

Mellanby also lays some stress on the supposed anti-infective action of vitamin A (now more generally agreed to be a function of vitamin C) and makes the following interesting suggestion: "Is it not possible . . . that the beneficial effect of the malarial treatment of general pa-

ralysis of the insane is due to the liberation of large quantities of vitamin A and other protective factors from the liver of the patient infected with the malarial parasite? It might be worth while before giving malarial treatment in such patients to fill their livers up with this vitamin by giving diets rich in this substance and cod-liver oil. It is possible that the uncertainty of this strange form of treatment would disappear if this factor were taken into consideration."

Three years later, Mellanby (1934)[140] described further interesting and pertinent experiments. During his investigations into the etiology of rickets in young dogs, he became interested in the incoördination of movements that often appeared in these animals. He felt that the two conditions that determined the onset of ataxia were, first, the absence from the diet of fat-soluble vitamins, and, second, the presence of a high cereal intake and especially a large amount of wheat embryo. In addition to incoördination these animals often appeared to be mentally affected; they seemed dazed and were incapable of fixing their attention upon anything for more than a moment. Another early sign to develop was spasticity, especially of the hind legs, which ultimately became very weak; cramps and convulsions also occasionally developed.

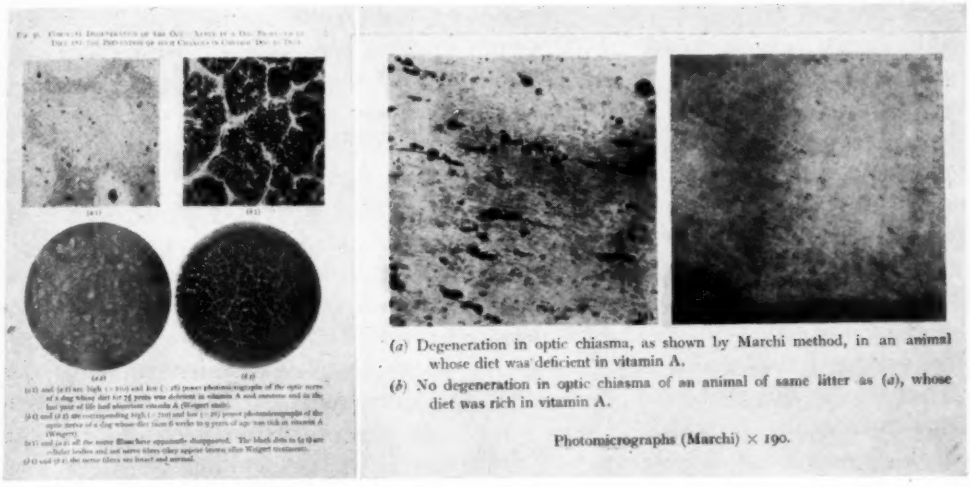
Mellanby felt that the nutritional deficiency responsible for this neurological picture was the vitamin-A portion of the fat-soluble complex and not vitamin D. The changes could be prevented if butter, egg-yolk, or cod-liver oil formed part of the diet, and also by carotene. If the incoördination were allowed to develop and vitamin A or carotene was subsequently added, the animals often improved very markedly. If, on the contrary, ergot was added to the diet these degenerative changes became much worse.

A similar clinical and pathologic picture of neurologic damage to the spinal cord was produced by Mellanby in young rabbits, rats, and birds with a diet deficient in vitamin A. It seems important to note that he was particularly successful in producing the pathologic abnormality in young animals.

Since the ataxia in his experimental animals was often greater than could be accounted for by the lesions found in the central nervous system, especially the spinal cord, Mellanby carried out in certain animals pathologic examinations of the vestibular division of the eighth nerve. This also showed demyelinization and degeneration, which, he felt, explained the excessive incoördination and abnormal head movements. Similar but less marked degeneration was also found in the cochlear division of the eighth nerve. Because of this finding, he was led to the examination of the peripheral nerves in general, and found that here also there was degeneration of the posterior roots and in the peripheral nerves. In at least two other cranial nerves, namely, the afferent nerves of the eye including the optic nerve, and the trigeminal nerve, there was the same type of demyelinization. On the basis of this experimental fact, Mellanby feels that it is possible that vitamin-A deficiency may play a part in some types of retrobulbar neuritis. In his experimental animals which showed demyelinization of the optic nerves, the ganglion cells of the retina also showed degenerative changes; when a vitamin-A deficient diet was given over a long period of time, the whole ganglion-cell layer of the retina occasionally disappeared and with it also the optic nerve, which was replaced by connective-tissue strands. There is reproduced herewith as figure 8 Mellanby's figure showing the optic nerve from a dog which was fed on a vitamin-A-deficient diet for seven

years; this dog became completely blind and showed complete degeneration of the optic nerve. For control a normal optic nerve is likewise shown.

Mellanby lays further stress on the fact that vitamin-A deficiency predisposes to infection. He feels that the predisposition to infection produced by vitamin-deficient diet is interrelated to the nerve degeneration that occurs under these circumstances. For example, when animals fed a diet deficient in vitamin A develop kera-



(*a*) Degeneration in optic chiasma, as shown by Marchi method, in an animal whose diet was deficient in vitamin A.

(*b*) No degeneration in optic chiasma of an animal of same litter as (*a*), whose diet was rich in vitamin A.

Photomicrographs (Marchi) × 190.

Fig. 8. Reproduced from Mellanby (reference 140).

tomalacia or xerophthalmia, examination of the ophthalmic branch of the trigeminal nerve showed degenerative lesions concomitant with the degree of inflammatory lesions in the eye. Mellanby points out that both xerophthalmia and night blindness in man are directly related to a diet deficient in vitamin A.

As to night blindness, he feels that the chemical defect in the retina may be secondary to retrobulbar changes in the optic nerve, and likewise to specific retinal changes in the nerve elements. He refers to the clinical reports of Moore concerning retrobulbar neuritis due to vitamin-A deficiency in Nigeria and says: "It is certainly true that in many cases of retrobulbar neuritis in man, other factors than vitamin A deficiency are responsible (*vide* disseminated sclerosis), but it is a practical point also worthy of consideration, whether in such cases vitamin A may not be of some etiologic or prophylactic or curative significance; I have in mind particularly other toxic amblyopias including the tobacco and alcoholic type."

In this 1934 report, Mellanby discusses the relationship of his experimental results on the production of degenerations in the nervous system by vitamin-A deficiency diet to certain clinical conditions observed in man such as beri-beri, convulsive ergotism, lathyrism, subacute combined degeneration of the spinal cord in pernicious anemia, and disseminated sclerosis. In this particular report, however, he has nothing to say on the subject of tabes dorsalis in general nor of optic atrophy in particular.

While both Mellanby, on experimental grounds, and Moore, on clinical grounds, seem reasonably certain that the spinal-cord damage and retrobulbar neuritis produced in animals or man is due to a diet deficient in vitamin A, the work of

subsequent investigators throws some doubt on this point. In 1934, Zimmerman and Burack[141] reported the production of essentially similar lesions in dogs fed on a diet deficient in vitamin B_2 (G). These workers used eight dogs, of which four were kept on an essentially normal diet as controls, and four were maintained on an artificial balanced ration adequate in all dietary essentials, so far as is known, except water-soluble heat-stable vitamin B_2 (G). The experimental animals developed, after a period of time ranging from 200 to 300 days, a slowly progressive disease characterized by loss of weight, persistent vomiting, diarrhea, and marked muscular weakness; all the animals died. The clinical features of this condition were quite different from those characterizing the canine disease known as black tongue. The anatomic changes consisted of marked demyelinization of the peripheral nerves, including the vagus, degeneration of the medullary sheaths, and replacement by gliosis of the posterior columns of the spinal cord; there were also degeneration of the medullary sheaths of the posterior, and less often of the anterior, nerve roots of the cord, and occasionally slight degenerative changes in some of the other fiber tracts. Zimmerman and Burack call attention to the fact that degenerative lesions of the central nervous system similar to or identical with these have frequently been described in pellagra in man. They also comment that the lesion produced in their dogs was so like the spinal-cord damage produced by tabes dorsalis in man that further studies are necessary.

In 1932 Barletta,[142] in a paper inacessible to us but quoted by Fazio,[143] reports that he found degeneration of the myelin sheaths of the nerves in pigeons with B avitaminosis. This is in contrast to the previous negative results of similar experiments performed by Riquier;[144] and also could not be confirmed by Fazio,[143] who examined the optic and sciatic nerves of 12 pigeons fed on a vitamin-B-deficient diet and was unable to find any changes in the optic nerves or chiasm, although there was degeneration of the myelin sheaths and swelling, varicosity, and tortuous course of the axis cylinders of the sciatic nerves.

The next report of the production of optic atrophy in animals by a presumably deficient diet is that of Moore, Huffman, and Duncan.[145] These authors observed 24 cases of complete blindness due to primary optic atrophy in calves developing shortly after birth and consequent on a diet of poor-quality roughage fed their mothers during pregnancy. The lesion also developed in young animals when poor-quality roughage was fed to themselves. In addition to blindness, there was often weakness, paralysis, and spasms; the pupils were dilated, there was no inflammation of the external coats. Pathologic examination showed that the lesion was due to optic atrophy with compression of the optic nerve where it passed through the bony foramen; this compression was apparently due to exostosis of the bone at the foramen. The condition was not observed in adults. The authors do not attempt to define the deficient component of the diet.

In 1937 Zimmerman and his associates carried their observation farther.[146] In these experiments the authors used 27 dogs, of which 12 served as various types of controls, 6 were fed diets that produced an acute untreated vitamin-G deficiency, and 9 a chronic vitamin-G deficiency. Of the 15 animals which received an inadequate amount of vitamin G, 12 showed definite neurologic lesions. The ration employed was adequate in all known dietary essentials except water-soluble heat-stable vitamin G. These 12 dogs slowly developed a progressive disease character-

ized by loss of weight, vomiting, bloody diarrhea, muscular weakness, incoördination, and decrease of the deep reflexes; death occurred in from 107 to 599 days. The acute symptoms could be modified and the life of the animal prolonged by the administration of a minimal amount of vitamin G. The anatomic lesions observed consisted of marked demyelinization of the peripheral nerves, and degeneration of their axis cylinders. Many medullary sheaths and axis cylinders of the posterior columns were also destroyed and replaced by gliosis. These degenerative changes of the nervous system are similar to those observed in cases of human pellagra. In this paper there is no mention of atrophy of the optic nerve or analogy of the neurologic lesions to tabes dorsalis.

Perhaps the most significant neurologic lesions produced in experimental animals by deficient diet are those described in a report by Wintrobe and his co-workers in 1938.[147] This is a preliminary communication dealing with the production of a neurologic disease in young pigs. The total number of experimental control animals is not stated. Young pigs were given an artificial, presumably adequate, diet in all respects. As they developed, the quantity of yeast originally provided to include the total vitamin-B complex was gradually reduced while thiamin (vitamin B_1) and riboflavin were given in its place. The rate of growth decreased, the general condition of the animals became impaired, and marked ataxia without motor weakness developed. Histologically there was severe degeneration of the posterior columns of the spinal cord, the dorsal-root ganglion cells, and peripheral nerves. The pathologic picture in the spinal cord was essentially identical with that of tabes dorsalis in man. There is no mention of the optic nerves in the paper by Wintrobe and his co-workers except the statement that the cerebrum was quite normal.

Lee and Sure[148] suggest that much of the conflicting evidence as to nerve degeneration in animals fed on various deficient diets may be due to the histopathologic technique employed. They studied various portions of the nervous system (sciatic, trigeminal, and optic nerves, and the spinal cord) in rats fed on diets deficient in vitamin B_1 (9 animals), whole B complex (38 animals), and vitamin A (20 animals), by a special technique described in their title. Degeneration of the trigeminal nerve was common to all groups, thus suggesting that Mellanby's explanation of the possible relationship of such degeneration to xerophthalmia in vitamin-A-deficient animals is not correct. Important to note, however, degeneration of the optic nerves was not observed in any of the 47 animals with B avitaminosis but did occur in 10 of the 20 (50 percent) of those fed on an A-deficient diet. The duration of the dietary deficiency was apparently not a factor, since, in those animals developing optic-nerve degeneration, the number of days on the deficient diet ranged from 35 to 68, averaging 51.8, while for those with no degeneration the range was from 14 to 65 days, averaging 48.5. Nor did involvement of the optic nerves parallel involvement of other parts of the nervous system.

Imachi and Takamasa[149] have approached the effect of vitamin deficiency on the optic nerves in an interesting manner. In three series of rats, one fed a normal, another a vitamin-A-poor, and the third a vitamin-B-poor diet, they studied the effect of various poisons and bacteria (carbon monoxide, carbon dioxide, and ammonia [NH_4], tubercle and colon bacilli, pneumococci, staphylococci, and streptococci) on the retina and optic nerves. All these substances, excepting ammonia, caused disturbances in the re-

tina and optic nerves of normal animals; in rats on a vitamin-A-deficient diet, the changes were much more pronounced, with striking degenerative changes in the nerves and inner cell layer of the retina; while in vitamin-B-deficient animals, hardly any alterations were present.

It is extremely difficult to interpret these contradictory findings in experimental animals. It seems reasonably clear, however, that with a diet deficient in vitamin A, vitamin B, or both, there may occur degeneration in the spinal cord, and sometimes in the optic nerves. When the diet is apparently deficient in vitamin A only, this degeneration is spotty in nature and is not sharply localized in the posterior columns of the cord. When the diet is apparently ample in vitamin A but deficient in some part of the B complex, the degeneration in the spinal cord is apparently sharply limited to the posterior columns and there is produced a pathologic lesion that is completely indistinguishable from that of tabes dorsalis in man. There is conflicting testimony as to the involvement of the optic nerves in both vitamin-A and -B deficiency, though it seems clear on the basis of the work of several observers that degeneration in these nerves may be produced by a deficient diet. The particular component or combination of components responsible for this neurologic degeneration in the spinal cord, and in the cranial nerves is not yet clear, and much further experimental work must be done before the results are applicable, if ever, to the human problem of tabes dorsalis. It is, however, strongly suggested by virtue of the clinical peculiarities of tabes dorsalis as compared to other forms of neurosyphilis repeatedly referred to above, and by virtue of the experimental work described here, that the pathogenesis of tabes and of primary optic atrophy may conceivably depend on a combination of neurosyphilis and dietary deficiency. It is likewise probable that the elucidation of this problem will depend on the laboratory rather than on the clinic. Information as to dietary deficiencies in tabetic patients is first of all extremely difficult to obtain, especially since the dietary deficiency, if any was present, must relate to a time period many years before the patient is first seen with tabes, while the lesions in the spinal cord and elsewhere are in the process of evolution. Moreover there is increasing evidence in the nutritional field that actual dietary deficiency may not be necessary for the production of certain of the deficiency diseases; instead, the patient may develop these diseases in spite of an ample diet by virtue of the fact that he fails to metabolize properly certain components in his diet. The field is a fertile one for further experimentation.

Summary

This paper critically examines the recent literature on the pathology and pathogenesis of syphilitic primary optic atrophy. The scanty pathologic data available throw little light on the pathogenesis of the condition, although they seem conclusively to demonstrate that optic atrophy is not due to actual syphilitic inflammation of the optic nerves dependent on the presence therein of the causative organism.

The newer theories as to the pathogenesis of optic atrophy are five: (1) It may be due to the virus of lymphogranuloma inguinale. This theory has so far nothing to support it. (2) It may be due to vascular constriction, functional or anatomic, within the optic nerves, with subsequent nutritional disturbances leading to atrophy; and it may be relieved or arrested by the use of vasodilator drugs. This theory seems ill-founded, and is now largely abandoned. (3) It may be due to a disturbance of the interrelation-

ship between the factors of intraocular tension and retinal arterial systolic and diastolic blood pressure (the latter dependent on systemic hypotonia). This theory, advanced by Lauber and Sobanski, seems unsupported by the observed facts. (4) It may be due to adhesive optochiasmal arachnoiditis. While this theory may explain the occasional case, it certainly does not suffice for many others in which arachnoiditis cannot be demonstrated at operation or necropsy. (5) It may be due to the combination of neurosyphilis and nutritional deficiency. This theory has some clinical and more experimental backing, but requires much further study before it can be accepted.

In short, it seems probable that the pathogenesis of syphilitic primary optic atrophy, so often (perhaps almost always) associated with tabes dorsalis, is identical with that of tabes dorsalis itself. This, in turn, is as yet unknown, though recent studies, especially in the field of nutrition, offer hope of its elucidation.

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GRENZ-RAY TREATMENT OF EXPERIMENTAL INFECTION OF THE CORNEA*

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The clinical value of Grenz rays in the treatment of inflammatory diseases of the cornea has been demonstrated by a number of European workers[1, 2, 3] and more recently by one of us (R.P.[4]). Grenz rays, which may be considered soft roentgen rays, are effective in the superficial tissues in which they are absorbed[5, 6] and are especially adapted, therefore, to the treatment of surface lesions of the eye.

Since staphylococci are frequently implicated in lesions of the cornea and experimental infections may be produced by them, it seemed desirable to determine whether the progress of such lesions could be affected by treatment with Grenz rays.**

METHODS

Rabbits of mixed breeds, weighing between 3,000 and 4,000 gm., were used. Staphylococcal injections were made intracorneally in both eyes and one eye treated, leaving the fellow eye as control.

Injection of staphylococci. A mannite-fermenting, coagulase-producing orange staphylococcus isolated from a case of panophthalmitis was used in all experiments. Fresh saline suspensions of 18-hour agar-slant cultures were made and standardized by turbidity measurements. Injections were given with gauge-27 needles and tuberculin syringes into the substantia propria of the cornea 4 to 5 mm. from the limbus. Pontocain 0.5 percent was dropped into the conjunctival sacs previous to the injections. The number of organisms injected varied somewhat with each experiment, as is noted in the protocols. Great care was taken to have the injections as similar as possible in treated and untreated eyes. The lesions were observed and measured at regular intervals over a period of several weeks.

Grenz-ray treatments. Before treatment both eyes were anesthetized with pontocain; the lids and nictitating membrane of the right eyes were retracted and as much as possible of the cornea exposed to the rays. Each treatment lasted one minute and consisted of 485 R with the following factors: 10 Ma, 11 Kv, and 8 to 9 cm. distance. The number and spacing of treatments is given for each experiment.

Experiment 1. Since we had found that local antibody stimulation in the cornea by Grenz rays was greatest with repeated doses previous to the injection of the antigen (see below), in the first experiment a series of irradiations was delivered previous to the corneal infections. Eight treatments were given on alternate days to the right eyes of four rabbits. Four days after the last treatment 0.03 c.c. of a suspension of the staphylococcus (approximately 9 million organisms) was injected into both corneae of all rabbits. The measurements of the resulting lesions recorded on chart 1 indicate that the Grenz rays did not affect the source

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** While this paper was in preparation McDonald and Pettit[7] reported that Grenz rays did not inhibit the development of staphylococcal ulcers produced experimentally in corneae of rabbits, but emphasized that their series was too small (5 eyes) and the dosages of Grenz rays used variable.

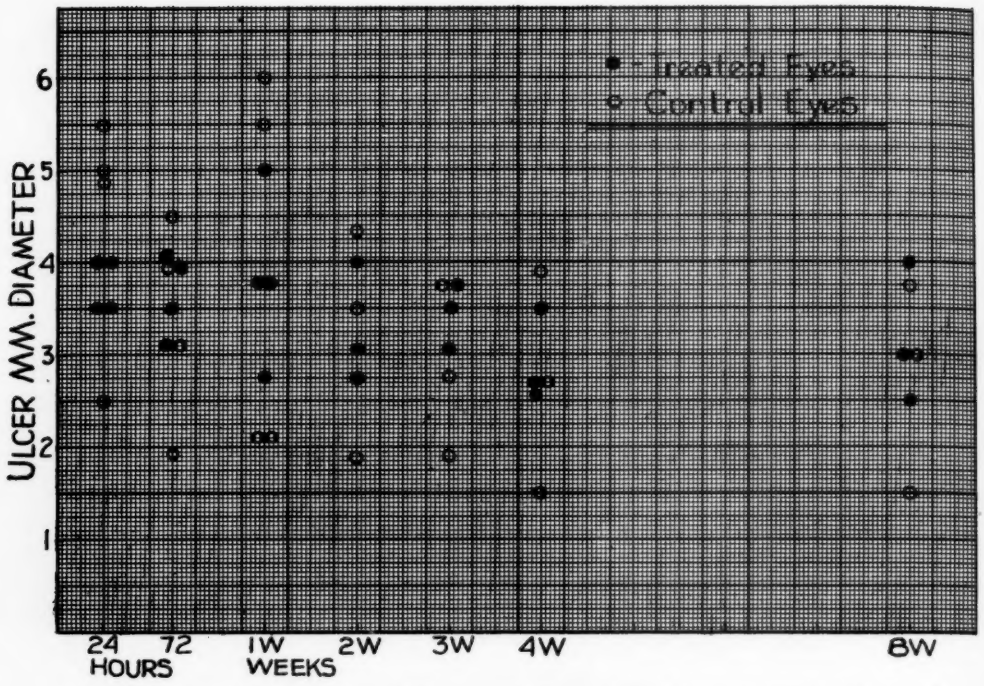

Chart 1 (Gallardo, Pfeiffer, and Thompson). Experiment 1, measurements of corneal lesions in treated and untreated eyes.

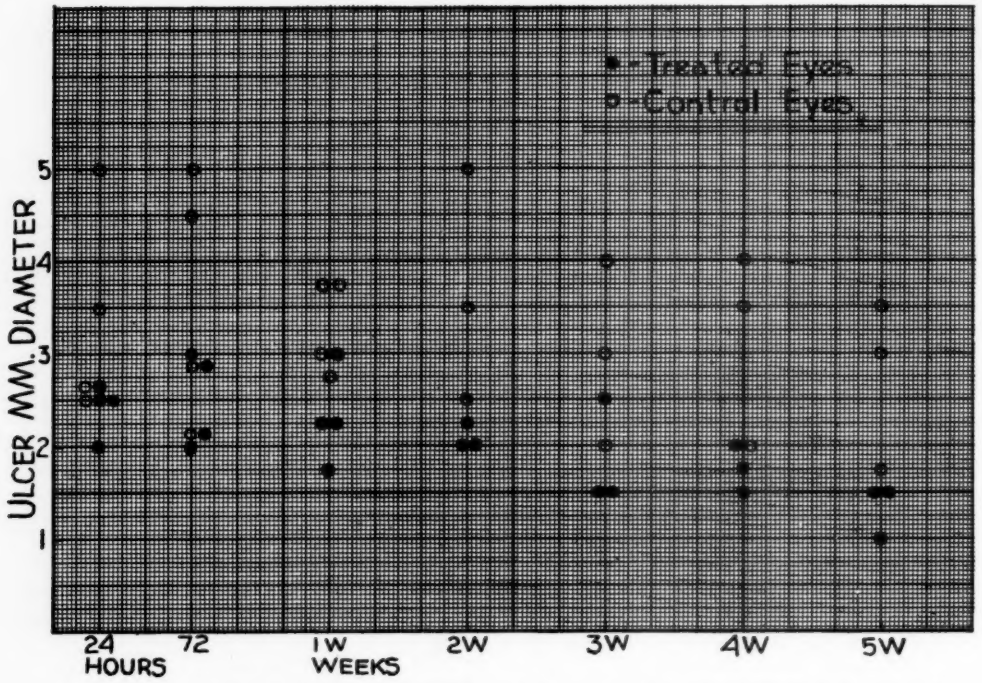

Chart 2 (Gallardo, Pfeiffer, and Thompson). Experiment 2, measurements of corneal lesions in treated and untreated eyes.

of the lesions. One rabbit died during the second week.

Experiment 2. Since treatments previous to infection had no effect, it was necessary to determine whether the rays would affect the course of the lesions when given after the infection. Both corneae of four rabbits were injected with 0.01 c.c. of a suspension of the staphylococcus (approximately 3 million organisms). Thirty minutes later all the right corneae were exposed for one minute to the rays. Thereafter the corneae were exposed every day until a total of six treatments had been given. The measurements of the resulting lesions are recorded on chart 2 (one rabbit died at the end of the first week). The number of eyes involved is small but it is evident that the ulcers and resulting scars were consistently smaller in the treated eyes. In addition hypopyon occurred in two control eyes, one of them with perforation of the cornea (Fig. 1 D). The anterior chamber in all treated eyes remained clear throughout the experiment.

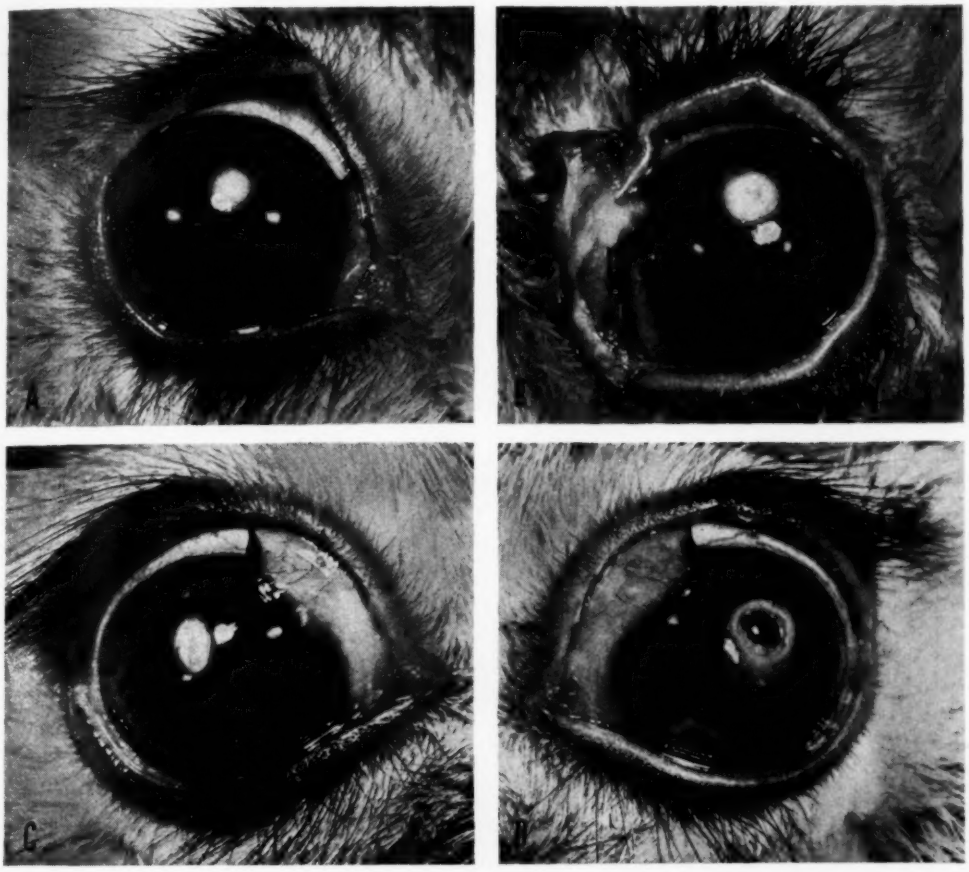

Fig. 1 (Gallardo, Pfeiffer, and Thompson). Experiment 2, eight days after infection; A and C show the treated corneae (OD's); B and D show the untreated corneae (OS's) on same rabbits.

Experiment 3. This was similar to experiment 2 except that six rabbits were used and more treatments given. Grenz rays were applied 30 minutes and 6 hours after the corneal injections of staphylococci and then daily for 6 days,

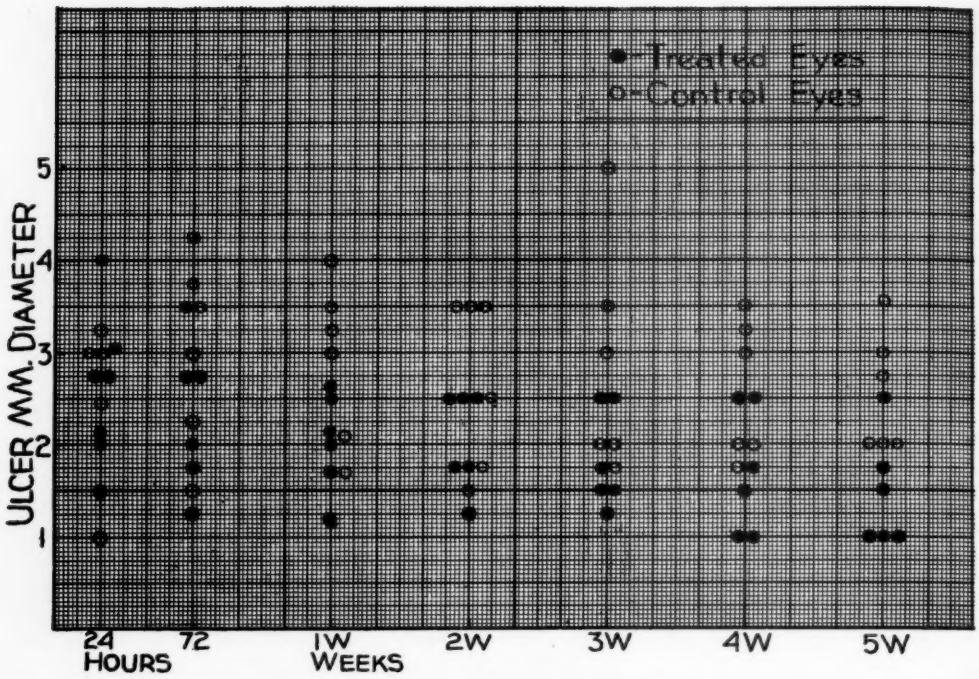

Chart 3 (Gallardo, Pfeiffer, and Thompson). Experiment 3, measurements of corneal lesions in treated and untreated eyes.

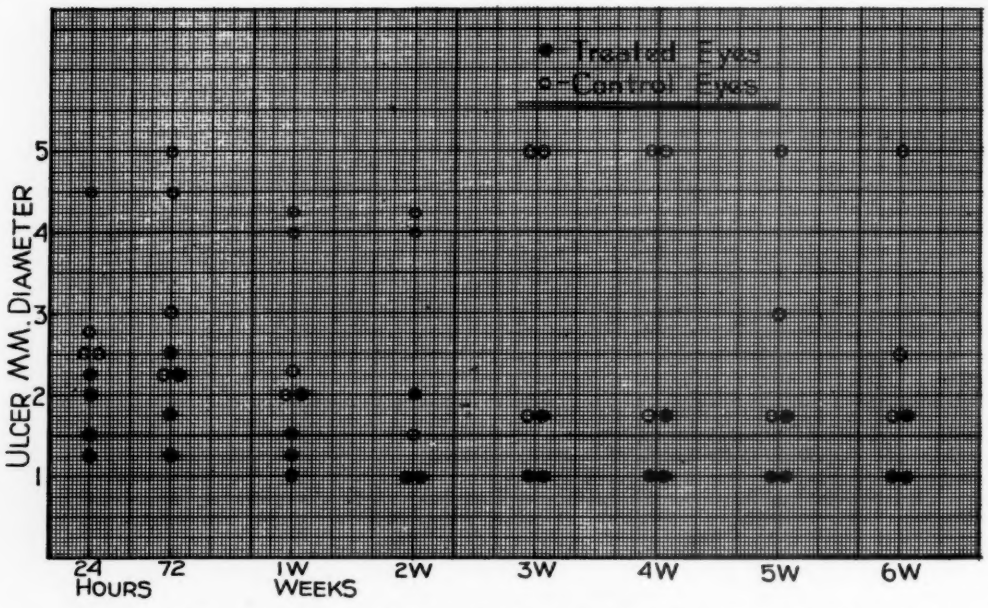

Chart 4 (Gallardo, Pfeiffer, and Thompson). Experiment 4, measurements of corneal lesions in treated and untreated eyes.

a total of 8 treatments. The results recorded on chart 3 show that the treatments inhibited the development of the ulcers; but in spite of the greater num-

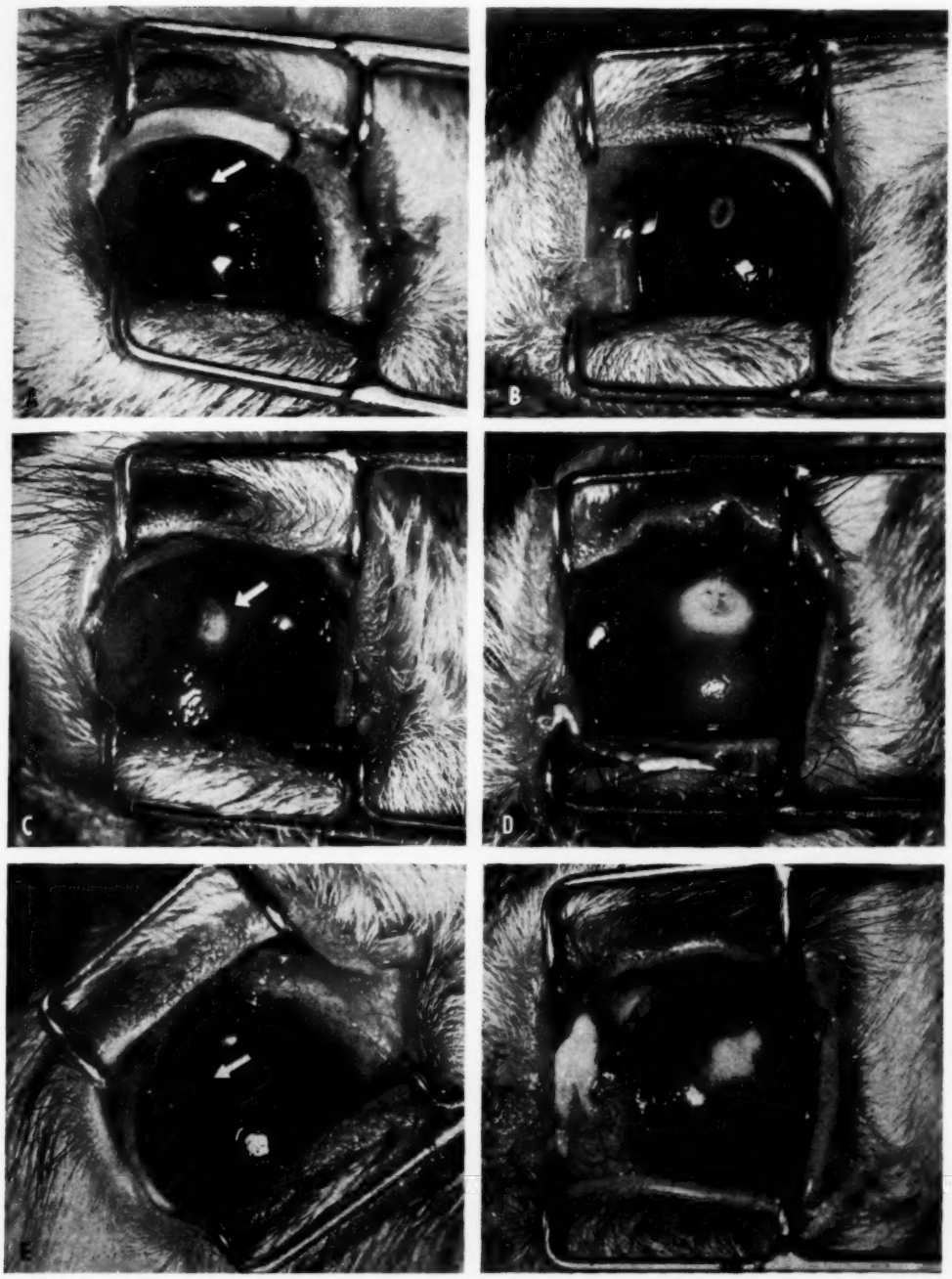

Fig. 2 (Gallardo, Pfeiffer, and Thompson). Experiment 4, 10 days after infection; A, C, and E show the treated corneae (OD's); B, D, and F show the untreated corneae (OS's) on the same rabbits.

ber of treatments and the more continuous application the results were less marked than in experiment 2. One control eye in this group developed pan-

ophthalmitis, none of the treated eyes did so.

Experiment 4. Four rabbits were given 0.02 c.c. of staphylococcal suspension (approximately 6 million organisms) by injection into both corneae. Grenz-ray treatments were given to the right corneae 30 minutes and 20 hours after the injections and then on alternate days until a total of seven treatments had been given. The results are shown on graph 4. One animal died after five days of the infection. Again the lesions in the treated eyes were consistently smaller than those in the controls (see photographs). One control eye developed panophthalmitis.

Summary and discussion

Lesions and resulting scars produced by intracorneal injections of staphylococci in rabbits were definitely and consistently smaller in eyes treated by repeated applications of Grenz rays following the infection than those in the untreated eyes of the same animals.

By combining experiments 2, 3, and 4 (12 treated eyes and 12 control eyes) and using the measurements at the 2-week period it is found that the average diameter of treated lesions was 1.87 mm., and of control lesions was 3.4 mm. The difference between the means is 1.57 and its standard error* is 0.355. The difference is four times its standard error.

Severe intraocular reactions (3 to 4 mm. hypopyon) occurred in two of the control eyes, in one of which the cornea perforated. Panophthalmitis developed in two other untreated eyes. In the treated eyes the intraocular reactions, when present, were always mild and brief and never developed into panophthalmitis. Conjunctival reaction was, as a rule, present in varying degrees in both treated and control animals but the purulent secretion disappeared more rapidly from the irradiated eyes.

The mechanism by which this form of radiant energy stimulates healing is not understood. In a previous paper[8] we reported that the local formation of precipitins following the injection of egg albumen into the rabbit's cornea was increased by exposure of the cornea to repeated dosages of Grenz rays. The effect was most marked when the exposures were made for several days prior to the injection of antigen. The failure to produce evidence of protection in the first experiment in which treatments were given in such a way as we had previously found to stimulate antibody formation in the cornea (that is for several days before the intracorneal injections) seems to indicate that the rays did not affect the lesions by the stimulation of antibodies against the organisms. In a few preliminary experiments we have found that the Grenz rays are bactericidal for staphylococci on the surface of agar plates although Bertrand[9] denied that they are bactericidal. Whether the Grenz rays are able to kill or injure the bacteria within the corneal tissue is a possibility yet unproved.

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* Calculated from the formula:

$$SE = \sqrt{\frac{\sigma_t^2}{N_t} + \frac{\sigma_c^2}{N_c}}$$

where $\sigma_t = \sqrt{\frac{\Sigma\delta_x^2}{N_t - 1}}$ $\sigma_c = \sqrt{\frac{\Sigma\delta_x^2}{N_c - 1}}$

and N_t = number of treated eyes
N_c = number of control eyes
δ_x = difierence between individual diameter and average of group.

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EVISCERATION OF THE GLOBE WITH SCLERAL IMPLANT AND PRESERVATION OF THE CORNEA*

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Removal of the eyeball from Tenon's capsule was originally proposed by J. M. Ferrall,[1] of Dublin, in 1841, who reported his first case with this operation in 1846. H. D. Noyes,[2] in 1873, advocated evisceration for panophthalmitis. In 1885 this procedure was modified by Mules,[3] who combined keratectomy with implantation of a hollow glass sphere in the sclera, in order to secure better cosmetic results. As was to be expected, Mules's chief difficulty lay in securing asepsis. A few years later Frost[4] and Lang[5] suggested an implantation in Tenon's capsule. Huizinga[6] performed a modification of this method, whereby, after keratectomy, he removed a larger section of the scleral cups posteriorly and performed an opticociliary neurectomy and implantation. No further reports of any modification of the Mules method are available.

Considerable controversy as to the relative merits of these two methods followed, and in 1896 a special committee was appointed by the Ophthalmological Society of the United Kingdom to "consider the relative value of simple excision of the eyeball and the operations which have been substituted for it." The committee[7] reported at length, and gave a historic review of the subject, referring to the incidence of meningitis and sympathetic ophthalmia and giving data regarding defective prominence and imperfect movement of the artificial eye, as well as asymmetric development of the face and orbit after removal of the eye in children. This committee summarized the advantages and disadvantages of each of the various substitute procedures for simple enucleation. In the light of modern technique this much-quoted, 73-page report appears to be quite valueless now. Of special interest, however, was a minority report by Thomas H. Bickerton, who disagreed with the conclusions of the committee. Question IV of the questionnaire sent out by the secretary, E. Treacher Collins, was: "What are the special advantages with relation to the wearing of an artificial eye?" referring to operations that were substitutes for simple enucleation. Bickerton was of the opinion that "when a diseased portion of the human body must be removed, the surgeon should not remove more than is absolutely necessary" and that, "in all cases of removal of the eye except (*a*) where sympathetic inflammation has already been excited, or (*b*) where the sclera is extensively lacerated or contused, Mules's operation, or simple evisceration,

* Read at the seventy-fifth annual meeting of the American Ophthalmological Society, The Homestead, Hot Springs, Virginia, on June 5, 1939.

is the rational operation on the general grounds before indicated." Bickerton stated further that, "as operation is often called for on cosmetic grounds alone, and excision inflicts a positive disfigurement, the radical operation insuring the best cosmetic result should be adopted. . . . Even where the sphere subsequently escapes, the case is converted into one of simple evisceration and a good support for an artificial eye remains."

We have well-defined indications for simple incision in panophthalmitis, for enucleation for intraocular malignancy, or for corneas badly damaged by injury with possible danger of infection, or when a specimen for pathologic study is required or very desirable. Simple evisceration is positively indicated for phthisis bulbi or painful blind eye, with implantation only if the eye is not too shrunken; in absolute glaucoma an implantation may be indicated for cosmetic reasons if the individual is not too old or too debilitated. There are many instances in which implantation of a sphere in a recently injured eye is desirable for purely cosmetic reasons to support the prosthesis.

The following statement is made by Spaeth[8] in the latest published book on ophthalmic surgery:[7] "Repeatedly operations have been devised of implanting a sphere of some type into the scleral shell. The reason given for these operations is that motion of the stump is thereby increased and the cosmetic appearance of the patient improved. Practically, the author [Spaeth] cannot concur in this. The postoperative course is never so uneventful, the reaction is more marked, the period of hospitalization is greatly increased, postoperative extrusion of the 'sphere' [whatever the sphere may be] is much more common, and the final stump is actually smaller than that obtained by other procedures. . . . Enucleation of the globe is the indication in practically all of the other reasons for the removal of the eyeball." It is impossible to accept or approve of this general condemnation of evisceration with implantation in the sclera. These objections are of slight significance in comparison with the great satisfaction attained by the excellent cosmetic results with scleral implantations and the improved motility afforded by retaining the original muscle insertions. From an abundant personal experience it has been found that practically all patients, and especially those who would otherwise hesitate to submit to removal of an eye, readily consent to evisceration with implantation. Since they desire the most nearly perfect cosmetic result obtainable, patients are willing to undergo the occasional greater reaction and slightly increased period of hospitalization that may be required to attain a freely movable prosthesis. If the clinical picture permits evisceration, and if age and general health do not constitute contraindications, the occupation and social status of most patients demand that operation which would inflict the least mutilation with the best possible cosmetic result. An intrascleral implantation is quite as simple and safe as one in the capsule of Tenon when either procedure is indicated and simple enucleation can be avoided. Implantations in Tenon's capsule by the Frost-Lang method have been frequently done by the writer in proper cases but were found less satisfactory than Mules's operation with preservation of the cornea, when possible, owing to the higher incidence of extruded spheres, displacement of the implant through the muscle cone, sinking in of the upper lid, and imperfect motility, regardless of the type of implant employed with the former procedure. Personal experience with the original Mules operation, with inclusion of a hollow, lead-free glass or gold sphere, corresponds exactly with that of

Moretti[9] and others, that approximately 25 percent of Mules's implants are followed by expulsion of the sphere. The frequency of loss of the implant, and the fact that many patients were encountered who are able to wear a prosthesis over a shrunken globe without irritation of the cornea, induced the belief that perhaps it was not necessary to sacrifice the cornea; that, by changing the suture line fewer implants would be lost and a better stump could be provided than with the method of Mules. The many instances permitting Mules's operation, when the cornea was not too severely damaged, induced the writer to modify the technique and experiment with implants with preservation of the cornea in selected cases. The loss of pathologic material and the bugbear of possible sympathetic uveitis gave rise to some hesitancy. The general impression that the Frost-Lang procedure is preferable to Mules's operation in preventing sympathetic involvement of the fellow eye hardly seems reasonable if, during evisceration, uveal tissue is thoroughly and completely removed. It is highly probable that reports of sympathetic uveitis following evisceration, both with and without implants, can logically be attributed to incomplete removal of the uveal tissue and defective sterilization of the scleral cavity. Even after old iridocyclitis, complete removal of all intraocular contents and disinfection of the cavity are as certain as is enucleation in the prevention of sympathetic uveitis. Although choroidal tissue may extend along the vortex or ciliary veins in their course through the sclera, the writer believes that removal of all visible pigment and intraocular disinfection minimize the danger of sympathetic uveitis. Thirty-five years' experience with simple evisceration with implants, both by the original method of Mules and according to the technique to be described, has not resulted in involvement of the fellow eye in a single instance.

It has been argued that evisceration results in diminished blood supply, causing degeneration and even calcification of the sclera, and that an irritable stump sometimes results that requires ultimate enucleation or sacrifice of the implant. This has never occurred in any case in the writer's experience. In general, it is good surgical practice to eviscerate and poor practice to enucleate an uninfected eye, except for intraocular tumor, when a pathologic specimen is desired, or when an injured eye is too severely damaged. Moreover, there are few contraindications to evisceration with implant, with preservation of the cornea, when the cornea is not diseased, and this is especially true in cases of recent injury.

Technically, the procedure recommended is quite simple. With retrobulbar novocaine injection, or under general anesthesia, a circumcorneal incision is made around two fifths of the cornea, and the conjunctiva reflected, leaving a margin of 5 mm. for closure adjacent to the cornea. A small scleral incision is made just anterior to one of the recti muscles, preferably the superior rectus, when possible. With one blade of a straight, blunt Stevens scissors, an incision is made between the uvea and sclera and extended to include about two fifths of the circumference. The ciliary body is separated with a spatula, an evisceration spoon inserted, and the intraocular contents separated and removed *in toto*. Using a blunt nasal speculum, hemorrhage is thoroughly controlled with compresses soaked in adrenalin, or by the application of a heated probe, mosquito forceps, or the application of a dull cautery to bleeding points. Hemorrhage must be completely controlled. The endothelium on Descemet's membrane is wiped off with a gauze applicator, and the scleral shell is freely irrigated, dried, and swabbed with 1-per-

cent iodine, neutralized after one minute with 5-percent cocaine, and again irrigated with saline or boric-acid solution. With Carter's introducer, a gold or lead-free glass ball (using one 18 mm. in diameter but sometimes one smaller or even slightly larger) is inserted. Further estimation for size with easy scleral coaptation is made. A 14-, 16-, or 20-mm. hollow sphere may be preferable, but in removal of the sphere one must avoid inflicting undue trauma and making traction on the optic nerve. The sphere should not fill the sclera too tightly and thereby place tension on the sutures. If it is too small it will fail to aid in the control of postoperative oozing within the sclera; nevertheless a slightly too-small sphere is preferable to one that is too large. Temporary sutures are placed in the exact ends of the scleral incision for lateral traction by the assistant, in order to secure perfect coaptation of the scleral margins. Fine, white-silk, interrupted sutures, usually about six in number, are mattressed through the sclera. The conjunctiva is closed with black twisted silk, which is removed after a week. A gauze-cotton compress pad is pressed over the eye with elastoplast and left in place for three days. It is again applied after each dressing.

During the healing stage, a moderately severe tenonitis with some protrusion of edematous conjunctiva may develop. If this occurs, as it does in about half the cases, vertical conjunctival incisions under topical anesthesia, with the application of a snug pad saturated with lead and opium lotion, will gradually effect its disappearance. After the first day, if the patient does not rotate the eyes, and keeps closed the eye that has not been operated on, pain will be slight. The reaction is less marked than after Mules's operation with removal of the cornea. Patients should remain in bed for three or four days. Hospitalization averaged 13 days for 22 patients.

At variable periods an interstitial vascularization of the cornea occurs until finally the cornea is completely supplied with blood vessels, a process that becomes complete in from three to six weeks. This is evidently a natural process to supply nourishment to the cornea. Complete opacity does not develop, however, and the implant remains visible. An artificial shell may be fitted after three weeks. Owing to the excellent nutrition of the cornea from the extensive vascularization, no ulceration occurs, and the shell prosthesis is worn with comfort. In only two instances have buried silk sutures given rise to trouble—once from a scleral incision made too near the cornea, when late erosion necessitated removal, but without separation of the scleral wound; in the other instance there was separation of the scleral wound with exposure of the gold ball. Infection did not occur in any case. In the case cited the sphere was removed and the globe collapsed, after the method of Harold Gifford. The writer has had no opportunity of seeing the result after an artificial eye was fitted in this patient. As a rule, private patients secure an especially made eye to match its fellow, but in general a very good-fitting eye of a color and pattern to match the fellow eye may be obtained from stock. It has been observed that divergent blind eyes require no muscular correction, and that binocular fixation appears to be perfect if the artificial-eye maker fits a proper prosthesis.

Summary

The cosmetic result and general comfort following evisceration without keratectomy exceed those obtained by the method of Mules. Irritation, lacrimation, and accumulation of secretion are less than after other methods. A sunken globe

or loss of the lid fold does not occur, and the appearance and motility usually defy recognition of the fact that a prosthesis is worn. Patients are pleased with the better cosmetic results obtained. There is relatively less reaction with the technique described than after Mules's operation, involving sacrifice of the cornea. In Mules's operation this sacrifice is unnecessary and possesses no advantage. The results in the series reported have been so satisfactory that further experience with the method described is warranted.

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RETINAL DETACHMENT OCCURRING IN PRIMARY COMPENSATED GLAUCOMA

Report of three cases

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The occurrence of retinal detachment in a preëxisting glaucoma is very infrequent and it was therefore considered worthwhile to report three cases that have come to our attention. These detachments occurred in eyes that had recently had elevated intraocular tensions; in two there was a definite diagnosis of primary compensated glaucoma, and in the third there was an elevated intraocular tension in an aphakic eye, the lens having been removed 35 years previously. The distinction must be made between the type of case here reported and that in which a secondary elevation of intraocular tension occurs at some time after the appearance of the retinal detachment. The latter type of case, while not common, is not so infrequent as the type referred to here.

Case reports

Case 1. Mrs. S. M., 50 years old, had been short sighted ever since she could remember. She was first seen on January 4, 1937, complaining of poor right vision which had been present for five days. The patient was in poor health, suffering from hyperthyroidism, cardiac complications, and a bronchitis, and was being treated by an internist. Vision in the right eye was 8/200 and could be corrected (−5.50 D. sph.) to 12/200; in the left eye it was 10/200, correctable (−6.50 D. sph.) to 10/70.

Inasmuch as we are here concerned with the left eye, it will suffice to say that there was a retinal detachment in the right eye for which she was operated on as soon as her general condition permitted. This operation, as well as a subsequent one, failed to achieve a good result; ultimately there was a complete funnel-shaped detachment in this eye.

The findings for the left eye on January 4, 1937, were as follows: the conjunctiva showed a moderate chronic inflammation. The cornea was clear, except for a Krukenberg spindle seen on slitlamp examination. There was no beam nor were there precipitates indicative of an iritis. The pupil was round, regular, and central, and reacted directly and consensually to light. The media were clear. The fundus showed a circumpapillary atrophy extending inferiorly (myopic) and there was marked choroidal sclerosis. It was thought that the fundus changes were sufficient to account for the relatively poor vision. The visual field was full.

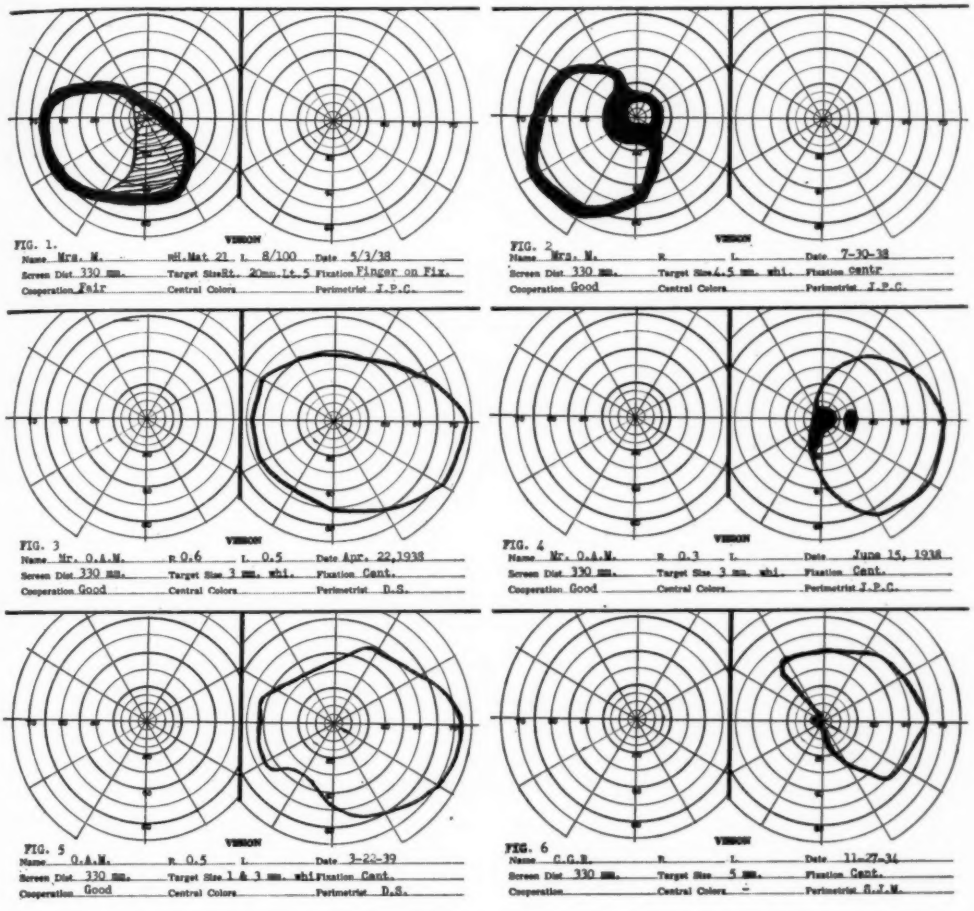
In September, 1937, the corrected vision of the left eye was 10/50. The objective findings were just as they had been nine months previously except that intraocular tension in the left eye was 33 mm. Hg (measured with a Gradle-Schiötz tonometer as were all subsequent tonometric recordings). The visual field was still full, showing a possible slight enlargement of the blind spot. The patient was put on 2-percent pilocarpine nitrate b.i.d.

The intraocular tension in the left eye remained between 20 and 30 mm. Hg for the next six months with no change in the objective findings nor in the visual fields. On March 30, 1938, in spite of the regular use of 2-percent pilocarpine b.i.d., the tension was 46 mm. Hg. Vision was 10/40 with −7.00 D. sph. ⌯ −1.50 D. cyl. ax. 30°. One-third percent eserine solution was added to the pilocarpine, and on

April 4, 1938, the tension was 27 mm. Hg.

On April 11th, the pupil of the left eye measured 3.5 mm., the tension was pathologically soft, and the vision corrected was less than 10/200. The patient was given an appointment to return for study and work-up, but did not return for over three weeks.

On May 3, 1938, the corrected vision of the left eye was 8/100. The slitlamp revealed the same findings as previously except that an occasional cell was noted in the anterior chamber; there was no beam nor were there precipitates. Study of the fundus revealed a massive retinal detachment inferiorly, extending forward in two billows. One hole was seen at the 6:00-o'clock position, far in the periphery, and there was a suspicious lesion in the macula suggestive of a hole. Transillumination was negative (fig. 1).



Figs. 1 to 6 (Gradle and Snydacker). Fields in three cases of retinal detachment with primary compensated glaucoma. Figs. 1 and 2, case 1; figs. 3, 4, and 5, case 2; fig. 6, case 3.

On May 16, 1938, a multiple diathermy puncture for retinal detachment was performed on the left eye.

On July 9, 1938, the corrected vision of the left eye was 10/70; tactile tension was soft, the pupil was 6 mm. in diameter, the vitreous rather murky, but the retina

was flat throughout, a fact confirmed by a full visual field.

On July 23, 1938, the patient was again seen in the office. Four days previously she has noted sudden loss of vision in the left eye, accompanied by electric flashes. Vision was found to be the ability to count fingers at two feet, nasally. The pupil was 6 mm. in diameter, and the tension was soft. The fundus revealed a massive detachment of the retina on the temporal side, with a definite hole at about the 6:00-o'clock position, approximately where it had previously been noted. Visual field is shown in figure 2.

The condition remained unchanged until August 11th, at which time the surgical procedure was repeated (multiple diathermy puncture). The retina, however, failed to reattach, the detachment remaining particularly high inferiorly. There was no change during the next four months, and on the last visit on December 17, 1938, the corrected vision of the left eye with a −2.00 D. sph. was 12/200.

Case 2. Mr. O. A. M. presented himself on April 22, 1938, with the following history: Dr. Gessler of Fort Wayne had made a diagnosis of glaucoma in both eyes. The patient had been put on eserine, once daily in the right eye and twice daily in the left eye. The blurring of vision which had been his only symptom, and which had led to the diagnosis, had disappeared. His general health was good.

Examination revealed vision in the right eye of 20/30 + 3, and in the left eye of 20/40 −3; corrected vision with a −0.50 D. sph. ⊂⊃ +1.00 D. cyl. ax. 135° was 20/20 in the right eye and with a −0.75 D. sph. ⊂⊃ +1.25 D. cyl. ax. 45° was 20/20 in the left eye; with a +2.25 D. sph. added to both eyes he could read Jaeger 1 easily. The right eye was pale, the conjunctival vessels, particularly at the limbus, were somewhat engorged. The cornea was normal; the anterior chamber of moderate depth, clear, and without cells. There was no pigment absorption. The pupil measured less than 2 mm. in diameter and responded slightly to light. The lens showed a rather irregular, fairly dense opacity of the anterior cortex, subcapsular and slightly eccentric; there was a slight opacity of the posterior cortex. The disc was flat, slightly recessed, but not pathological. The vessels were in good condition and the fundus seemed normal. The left eye was similar to the right externally with the same anterior-lens opacity, but the lens of the left eye was otherwise clear. There was a definite pathological excavation of the disc in this eye, with a marked bend of the vessels, both above and below. There was a small glaucomatous halo. Otherwise the fundus was normal. Gonioscopy of the right eye revealed the chamber angle to be nearly closed with extensive anterior adhesions on the nasal side, less so above and below. Gonioscopy of the left eye showed the chamber angle to be slightly more open than the right, but there were extensive anterior adhesions. Peripheral visual fields showed extensive changes in the left eye, but the right appeared to be essentially normal. Intraocular tension was 42 mm. Hg in the right eye and 37 mm. in the left (fig. 3). The diagnosis of bilateral primary compensated glaucoma was established. Inasmuch as it is the right eye which interests us and the left eye has no bearing on the condition in question, the latter will be disregarded.

On June 2, 1938, the corrected vision in the right eye was 20/20 and the tension was 17 mm. Hg. The patient had been using a 2-percent pilocarpine nitrate solution q.i.d. and 0.5-percent eserine ointment at bedtime.

On June 15th the patient stated that four days previously he had had blue

vision and flashes of light in the right eye associated with blurring of vision. Corrected vision was 20/100 and the tension was 17 mm. Hg. The pupil could not be sufficiently dilated at this time to obtain a good fundus view; the visual field showed marked changes (fig. 4). The patient was instructed not to use any miotics for 24 hours and to return for another attempt at adrenalin dilatation. The next day the tension was 17 mm. Hg before an adrenalin pack was put in and 18 mm. afterward. The pupil dilated irregularly to 4 mm. in diameter and it could be seen that the entire temporal one half of the retina from the 12:00-o'clock to almost the 6:00-o'clock position was detached, being elevated 12 to 14 D. in the periphery and graduating down towards the disc. There were some folds of retina, but no holes were to be seen. The anterior chamber was negative. The patient was told to rest at home in bed, and was given 2-percent pilocarpine to use in both eyes q.i.d. On June 24th, the corrected vision was 20/50 and 20/30; the tension was: right, 22, and left, 30; but the pupil did not dilate sufficiently with an 1:1,000 adrenalin pack to permit accurate fundus study. Perimetry showed a marked improvement in the visual field. Three days later, in the hospital, under careful observation after a tension curve had been run, the pupil of the right eye was dilated with Links-Glaukosan, and the retina was found to be flat throughout. The picture did not change until August 22d, as the visual field shows, five days after the patient had been admitted to the hospital for tension-relieving surgery of the left eye. At this time there was noted a detachment of the retina of the right eye in the entire temporal one half, extending from the 11:00-o'clock to the 6:30-o'clock position elevated 12 D., and reaching to within 3 to 4 P. D. from the disc. The patient was sent home from the hospital for 10 days of absolute bed rest.

On August 30th there was a slight elevation of the extreme temporal periphery of the retina with a slight corresponding contraction of the visual field. On December 12th, the corrected vision of the right eye, was 12/200, and there was again an extensive bullous detachment of the temporal part of the retina. On December 21st, after five days of absolute bed rest, there was no change, so a multiple-diathermy-puncture operation for retinal detachment was performed. There was an uneventful postoperative course and a note was made on December 21st that the retina of the right eye appeared to be flat, which was the condition on discharge from the hospital on January 13, 1939.

On February 23, 1939, the retina of the right eye was flat throughout, the tension was 11 mm. Hg, and the corrected vision was 20/40. The visual field showed slight contraction on the nasal side (fig. 5).

On March 22, 1939, there was no change. The tension was 17 mm. Hg, and the visual field was slightly more full.

Case 3. C. G. R., a 42-year-old white male, presented himself on November 27, 1934, with the following history: His right eye had been operated on for congenital cataract at the age of three years, and his left had been operated on for the same reason at the age of seven years. When he was 11 years old he had been struck in the left eye by a baseball, and this eye had been subsequently enucleated. At the age of 34 years there had been an iritis in the right eye of unknown etiology which had cleared up satisfactorily under treatment. On November 3d, a little more than three weeks before his first visit, the patient had noticed a decrease in vision following the appearance of halos around lights for a period of one day. These halos had disappeared spontaneously and the vision returned to normal.

Two weeks later, after several days of prolonged close use, the halos reappeared and had persisted until the present time; there had also been a definite loss of vision, particularly on the nasal side, according to the patient's own statement. On November 22d, the tension had been 30 mm. Hg according to Dr. Connelly of Terre Haute who referred the patient, and pilocarpine had been given, to be used every four hours. With a +5.50 D. sph. ⊂⊃ +1.50 D. cyl. ax. 90° vision was 20/30 −3.

Upon examination of the right eye, it was found to be pale, the scleral veins slightly injected, the anterior chamber deep, the pupil slightly oval with the apex directed downward, and 2.5 mm. in diameter. There was slight iridodonesis and a moderate ectropion of the uveal pigment. The slitlamp confirmed these findings and revealed no new ones except that the oval shape of the pupil was due to a strand of vitreous extending forward. There was no visible beam nor were there any cells in the anterior chamber. Ophthalmoscopy revealed the disc to be shallowly excavated with a bending of the vessels at the superior and inferior margins that was suspicious. Temporal to the disc, there was a faint disturbance of the choroidal pigment, but the periphery of the fundus, as well as could be seen, was normal. The intraocular tension was 57 mm. Hg, and the field showed marked contraction on the nasal side (fig. 6). The patient was given a 2-percent pilocarpine-nitrate solution combined with a 0.5-percent eserine solution to use every four hours. The next day the tension had been reduced to 17 mm., the vision corrected was 20/30, and the patient was instructed to use his medication four times daily. On November 30th, he returned, and it was found that he was suffering a corneal erosion, the result of the tonometry. He was sent to the hospital for treatment, and the erosion cleared up quickly, but unfortunately, of course no tensions could be taken. Tactile tension remained normal.

On December 4th, while he was still in the hospital, there was a sudden marked loss of vision. The cornea was clear, the anterior chamber was clear, the pupil was 2.5 mm. in diameter and rigid. The fundus revealed an extensive detachment extending from the 7:30-o'clock to the 2:30-o'clock position, involving the entire upper three fourths of the retina including the macula. No tears could be located. After absolute bed rest for four days the retina flattened out considerably, so that only in the extreme temporal periphery was a detachment made out. On December 9th, a multiple-diathermy-puncture operation for retinal detachment was performed. The punctures included an area slightly larger than the original detachment because of the fact that no hole had ever been found. While the patient was still in the hospital, the retina again became detached, and further surgery was indicated.

On January 2, 1935, a modified Gonin operation with cautery point was performed. There was an uneventful postoperative record.

On March 4, 1935, the next note on the record states that the retina appeared flat and the tension as normal. The field showed some constriction above.

On April 1st, the field was much more constricted and the retina was definitely detached in the upper half from the 9:00-o'clock to 1:30-o'clock position.

On May 27th, the vitreous was clear; the entire upper half of the retina was detached, showing solid folds. Immediately below the horizontal meridian, the retina seemed flat, but was detached far in the inferior periphery. No holes were seen. Corrected vision with +8.00 D. sph. was 20/100.

On July 8th the condition was essentially the same except that the field

showed considerable contraction and the patient was sent to the hospital for further surgery.

On July 10th, a third operation was performed, with the use of superficial diathermy cauterization after the technique of Weve, covering the entire upper half of the globe. The convalescence from the surgery was complicated by a rise in intraocular tension that was controlled by miotics within 24 hours. On August 1st the eye was in order, but the pupil was so small that no fundus view could be obtained. The tension was normal. On October 21st, the eye was externally normal, and the tension was normal, but the fundus showed an extensive detachment, present in all quadrants, elevated to about 4 D. The corrected vision was 10/200. The condition objectively remained unchanged for a period of a year, but the field gradually failed.

On October 14, 1936, the patient was found to have definite slitlamp evidence of a uveitis in the form of a beam and cells, and, in addition, the intraocular tension was elevated to 50 mm. Hg. The patient was sent home on 2-percent pilocarpine and calcium gluconate twice weekly for three weeks. On his return on November 16th, the intraocular tension was 40 mm. and the uveitis that had been present had subsided somewhat, but his visual field had suddenly failed markedly. On November 25th, an iridectomy was performed with the dual purpose of lowering the tension and permitting a better fundus view. There was an uneventful postoperative course and on January 11, 1937, the corrected vision was 5/200 and the following note was made: Eye pale, pupillary area clear. Coloboma in order. The fundus shows no change, being detached in all quadrants. No holes are visible.

On January 13, 1937, the eye was again operated on for retinal detachment, this time with galvanism, after the method of Vogt. The patient had an uneventful postoperative course and was sent home. When seen on April 5th he had had a spontaneous vitreous hemorrhage three weeks previously which prevented any fundus view at this time. The tension was normal. Vision had decreased to light perception with questionable projection.

On May 19th, the retina was flat throughout, with scars from the many previous punctures clearly visible. The tension was very soft to the fingers, and the vision was reduced to doubtful light perception.

Summary of cases

Case 1: A mild glaucoma was discovered in a myopic eye and treated with a solution of 2-percent pilocarpine, to which a one-third-percent eserine solution was later added. After controlling the tension with these for some months a spontaneous retinal detachment occurred, which, after some delay, was successfully operated upon. The retina remained flat for about two months and then redetached, and a second operation proved unavailing.

Case 2: A diagnosis of a rather severe bilateral compensated glaucoma was established. A solution of 2-percent pilocarpine with 0.5-percent eserine to use at bedtime controlled the tension for a period of six weeks, when there was a spontaneous retinal detachment. This responded to bed rest, but redetached again and again responded to bed rest only. The third time the detachment occurred, however, it did not respond and surgery was resorted to with a good result.

Case 3: What probably was a secondary glaucoma developed in an aphakic eye. After using a 2-percent pilocarpine solution combined with a 0.5-percent eserine solution, which controlled the tension for a week, a retinal detachment developed that did not respond to surgical procedures. The glaucoma persisted at ir-

regular intervals after the detachment had occurred.

Review of these cases reveals that there are some factors common to all three. In the first place, they all had definitely elevated intraocular tensions at some period before the occurrence of the retinal detachment; in cases 1 and 2 the diagnosis was a primary compensated glaucoma and in case 3 a secondary glaucoma. In all three cases the detachment did not occur until after the tension had been reduced by miotics, and in all three cases the miotics used were a combination of pilocarpine and eserine.

Retinal detachment occurring in a preexisting glaucoma is an exceedingly rare complication. The explanation for its infrequency in cases in which the tension is elevated would seem to be fairly obvious; that is, the elevated pressure, spread evenly throughout the vitreous, tends to plaster the retina evenly against the underlying tissues, holding it firmly in position. The rarity of its occurrence in those cases of glaucoma in which the tension has been reduced, either by the use of miotics or by surgical intervention, is not so obvious. It was felt that if an adequate reason could be established why detachments did not commonly occur in such cases, it might be a step forward in determining the pathogenesis of idiopathic retinal detachment.

Theories as to the pathogenesis of idiopathic retinal detachment can be roughly divided into two classes; namely, (1) those theories which hold that the primary pathology is in the retina, and (2) those theories which ascribe to the vitreous a major role. The chief modern proponents of the theories falling in the first group are Vogt and Weve, who, though their views differ somewhat, believe that cystic degeneration of the retina is responsible for the formation of holes, and that the detachments result after the hole has been formed. Hanssen ascribed dehiscences in the retina to myopic stretching. Congenital weakness has been mentioned as a cause, particularly in those cases in which detachment with disinsertion of the ora serrata develops as the result of mild trauma. In the second group, those theorists who consign the primary pathology to the vitreous, are Gonin, Lindner, and Arruga, who, with relatively minor differences, all believe that the traction of a degenerated or detached vitreous is responsible for the origin of tears in the retina and the subsequent detachment.

It is almost universally agreed now that no one theory as to the pathogenesis of retinal detachment is operative in all cases, and that in some cases one or the other factor may be responsible, and in other cases a combination of factors may be causative. It was considered worthwhile, however, to study glaucoma cases in the endeavor to find out which theories might have been applicable in the cases here reported. It is a well-known fact that in sections the retina in cases of glaucoma shows marked degenerative changes. Marked cystic changes and local areas of chorioretinitic adhesions are frequently seen. On the other hand, there is no evidence as to the condition of the vitreous in cases of either primary or secondary glaucoma. Lindner, who has published the majority of the work dealing with clinical investigation of the posterior vitreous, implies that the vitreous in cases of glaucoma is normal, although he does not mention any specific work undertaken to prove his position. A study of the vitreous in cases of glaucoma was attempted in the hope that some light might be thrown on this subject. Unfortunately the technical difficulties encountered proved too difficult to make the study of definite value. The percentage of glaucoma cases in which the pupils

are, or can be, safely dilated to study the vitreous with the modified corneal microscope and the flat Koeppe contact glass are too few to make adequate observations. As a result of these clinical studies, no definite statement can be made as to the condition of the vitreous in cases of glaucoma which would throw any light on the cases reported here.

It is not inconceivable that a mixture of pilocarpine and eserine, exerting a powerful traction on the insertions of the ciliary body posteriorly at the ora serrata and anteriorly at the scleral spur by causing a spasm of the ciliary muscle, might cause a disinsertion of the retina at the ora serrata. However, this combination has been used many times in many other cases without producing such disastrous results. Over a period of five years since the occurrence of the first case in this series, 447 glaucoma cases have been seen by us. Of this number, 26 have received or are receiving a combination of pilocarpine and eserine, without deleterious effects. It would seem that there are additional factors acting in these three cases to produce the retinal detachment.

In reviewing the literature, it is surprising how few cases of the type here reported can be found. Vogt, in his textbook, mentions three cases, two of his own and one of Marchesani's, but does not discuss them nor attempt to explain the pathogenesis. Wilder reported a case in 1930 of a bilateral detachment occurring in a case of bilateral secondary glaucoma, while the tension was still elevated in both eyes. Bilateral iridencleisis not only reduced the tension, but left both retinae reattached. Dr. Wilder felt that the detachment might have been caused by a pouring out of choroidal exudate into the subretinal space as the result of irritative factors acting on the choroid. Through the courtesy of Dr. T. D. Allen, I can state that the patient's condition remains unchanged after nearly nine years, the tension remaining low, the retinae flat, and the peripheral visual fields full. Scott in England reported one case of bilateral glaucoma in which one eye developed a retinal detachment after an attack of herpes zoster ophthalmicus; the data supplied in this case unfortunately do not seem adequate, and there is no discussion.

Conclusions

1. Three cases of retinal detachment occurring in glaucoma in which the intraocular tension had been lowered by miotics are reported.

2. Review of the literature reveals the rarity of this complication.

3. The theories of retinal detachment are reviewed and an effort is made to correlate their importance in these cases.

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COSMETIC CORRECTION OF FACIAL ASYMMETRY WITH PRISMS*

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The case herewith reported demonstrates the end result of extended radium treatment for hemangioma of the skin of the temple and lip and malformation of the face with asymmetrical position of the eye on the affected side, for which cosmetic improvement of the low eye by means of prisms is recommended.

The patient, a girl 14 years of age, was seen in the Mandel Clinic Eye Department because of poor vision in the left eye and esotropia, for which she desired correction. She stated that the eye had turned in since earliest childhood when she had been sent to the Radium Association by the Infant Welfare Department of the Children's Memorial Hospital for treatment for a large birthmark of the left temple and left upper lip.

The patient was a full-term normal baby, normal delivery. She received extended treatment by a Chicago radiologist from her sixth week to her second year. He reported by personal communication that the hemangioma extended over the side of the temple, was elevated one-eighth inch above the skin surface, and formed such a mass on the left upper lid that the latter was constantly held shut and the child had to throw the head far back to see out of the left eye. The extent and size of the purple tumor led several of the consulting physicians to suspect a hemangiosarcoma. The radiologist reported that the patient received topical treatments of filtered gamma-ray radiation of radium over a two-year period, with irregular regressions of the tumor. At times the mass appeared to regress in one portion and relight into activity in another. The eyes and face were protected by lead shields but no prostheses were used. The radiologist had followed the patient at intervals up to the time when she came under the present writer's care (December 10, 1938).

During that time her only illness had been measles at three years of age. An early positive Mantoux reaction led to pediatric examination; normal general and chest findings, both physical and roentgenographic were reported. The blood Wasserman test was negative (August 8, 1936). She has one older brother six years her senior, living and well. Her parents are both in good health and neither report any familial abnormalities or growth anomalies.

Examination, December 10, 1938: Vision R.E. was 0.9-1. Under homatropine refraction with a -0.50 D. sph. $\bigcirc$ -0.25 D. cyl. ax. 120° it was 1.2; with the left eye it served to count fingers at 2 ft. (nas. eccentr.) unimproved by correction -0.75 D. sph. $\bigcirc$ $+$ 175 D. cyl. ax. 105° idim. Light perception and projection were good; the fields for white and colors accurate.

The left eye is 8 mm. lower than the right and the patient appears to hold the head somewhat inclined toward her right shoulder. The mouth and ear levels are symmetrical. There is an extensive 75-by 75-mm. scar over the left temple, involving the temporal half of the left upper lid down to the creases of the left external canthus. The cilia in this position are totally absent and the tarsus cannot be demonstrated on everting the lid. There is a denser scar of 18 by 18 mm. below the left nares, involving the left

* Presented before the Chicago Ophthalmological Society on May 8, 1939.

half of the upper lip up to the mucosal line, appearing like a repaired hare-lip but, from the history, an area of radiated hemangioma. Rhinologists report intact septum, and palate, and no evidence of congenital nasal abnormalities.

The strabismus angle of the left eye with and without correction measured with strabismometer, is 2 degrees of exotropia for distance, 1 degree of esotropia for near fixation with a +4-degree angle gamma in the fixating eye. The ocular motility is free throughout with some limitation in the direction of the left superior and lateral rectus muscles. Relative convergence is well performed (25 mm.).

The interpalpebral fissures measure R = 8.5 mm. L = 7.5 mm. with no impairment of lid motility. There is moderate but equal exophthalmos of 21.5 mm. in either eye. The corneal diameters measure 11 by 11.5 mm. in either eye. The conjunctivae are normal in each eye; there are no symblephara nor scarring. The pupil of the right eye measures 4 mm., the left 3 mm., with good light and accommodation reflex activity. The iris of the right eye is greenish-yellow in color; the left green with brown markings. Slit-lamp study further reveals neither pigment sprinkling, iris atrophy, nor lens opacities in either eye.

The disc and fundus of the right eye are normal. The macula of the left eye shows irregular peppering of pigment with intervening areas of depigmentation not seen in the right eye. The disc color, excavations, and borders are normal.

The visual fields measured with a Zeiss 33-cm. projection perimeter (7 ft-c), for 1 mm. white target and 3 mm. red and blue targets were normal for the right eye; for the left, the fields for the same targets were concentrically contracted 15 degrees, with absolute central scotomata for 3 mm. white and colors. Fields with the stereocampimeter confirmed the finding of an extensive left cecocentral scotoma.

Orthoptic measures of occlusion and binocular vision stimulation at the Mandel Eye Clinic proved to be of no avail (April 19, 1937, to November 22, 1937),

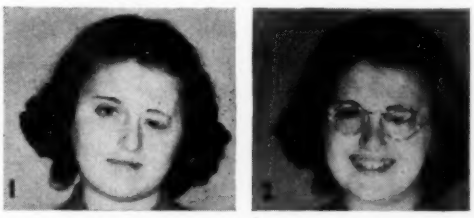

Fig. 1 (Cowen). Asymmetry and extensive post-irradiation scar of left temporal region.
Fig. 2 (Cowen). Correction of asymmetrical deformity with 20Δ prism, base down and in, at axis 150° before left eye.

and on April 30, 1938, in an attempt to make a cosmetic correction, the plan was conceived to place a prism, base down and in, to compensate for the low and esotropic position of the left eye. A prism of 20Δ placed with base at axis 150° was ground and fitted into the patient's frame. This made a much better appearance, provoked no diplopia because of the left amblyopia, and minimized the appearance of the extensive radiation scar. Figures 1 and 2 show the appearance of the patient with and without glasses.

The nature of the fundus lesion apparently is of an independent etiology and presumably not related to the radiation. The amblyopia of the left eye may perhaps be attributed to a central retinitic change that has crippled macular function, leaving only minimal pigmentation changes. Or the field changes may represent the result of prolonged suppression performed to neutralize an insuperable vertical diplopia.

The comment on the case concerns first surgical correction of the residual scar of the healed angioma. Although plastic

surgery has been recommended, it is felt that the rather heavy lens hides the most visible portion of the scar involving the upper lid and external canthus. The question of better appearance of the correcting lens before the left eye is pertinent: the optician has been approached concerning further rounding of the edges of the prisms. He states that this is a difficult and expensive job. As a further solution of this problem, one might consider the use of a strong cataract lens displaced down and in to give prism action, base in and down.

In summary, the condition of a girl 14 years of age is reported, who came for correction of an esotropia and lower position of the eye on the affected side. This eye is amblyopic, the iris in it is differently colored from its mate, and ocular motility is limited in the direction of its superior and lateral rectus muscles. Over the temporal region and left upper lid and upper lip on the left side is an extensive scar, the result of prolonged irradiation of a hemangioma treated in infancy. Cosmetic improvement by a 20$^{\Delta}$ prism placed base down and in (base across axis 150 degrees) is suggested.

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STUDIES ON THE INFECTIVITY OF TRACHOMA

IX. Immunological aspects of the rickettsial concept*

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During earlier studies on the etiology of trachoma, an abortive attempt[1] was made to determine the possible implication of rickettsiae in the evolution of the disease. The experiments undertaken at that time were subsequently discontinued primarily because (1) no structures simulating rickettsiae were detected in tissues from patients or animals experimentally infected; (2) tissue-culture experiments were completely unsuccessful in propagating the infectious agent; and (3) the Weil-Felix reaction was more or less consistently negative in the small series of patients observed. There were, secondarily, as expressed elsewhere,[1] essentially theoretical reasons, also, for discarding this approach, in favor of what has since appeared to be a more profitable study.[2, 3]

In the interim, however, a number of publications, stimulated originally by Busacca,[4] have affirmed and reaffirmed the concept that trachoma is a disease of rickettsial origin. While part of the supportive evidence for this belief is essentially morphological,[5] some is derived from tissue cultures,[6] and the remainder is dependent upon the Weil-Felix reaction.[7] While certain investigators are opposed to this concept,[8] it nevertheless seemed desirable to elaborate upon the earlier experiments undertaken in this laboratory regarding this possible relationship. In pursuing the present study, no particular emphasis was laid on morphological or tissue-culture analysis. The large number of smears and tissues examined routinely from patients and animals have always been devoid of rickettsial forms, and the repeated failure to propagate the infectious agent of trachoma by a number of modifications[9] indicated that until radical innovations were introduced further experiments with this

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technique would be unproductive. Consequently the burden of proof has been sought by immunological procedures, centering particularly around the Weil-Felix reaction, since it has occupied so prominent a position in the advocation of the rickettsial nature of trachoma.

Methods

Preparation of antisera. In order to supplement experiments with the sera of patients and animals suffering from trachoma, antisera were prepared with tissues scraped from patients during grattage. Both rabbits and monkeys, the latter of verified susceptibility to infection, were given repeated intravenous injections of triturated grattaged material. The injections were made every three to five days over a period of two months with tissues freshly obtained each time from different patients with clinically active trachoma. This method has already been described in a recent communication.[10] In addition, rabbits were immunized to Proteus bacillus, OX_{19}, by intravenous injections of formalinized, young (12 to 16 hours) broth cultures, administered on three successive days each week for four weeks. All animals were then bled 10 days following the final injection, and their sera were utilized in the experiments to be described below.

Technique of agglutination. Tests for the presence of agglutinins were conducted with sera from patients, or monkeys infected experimentally, and, in a few cases, with the sera from rabbits serving for intratesticular passage or purification of the infectious agent as described in a preceding publication.[11] Agglutinations were also done with the sera of animals immunized artificially as outlined above. For this purpose, suspensions of living bacteria (12- to 16-hour broth cultures of Proteus bacillus, OX_{19} and OX_2) were used, and incubation was accomplished at 42°C. and 37°C. for four hours, after which the tests were removed to the refrigerator (±6°C.) until the following morning when final readings were recorded.

Technique of neutralization. Equal quantities of undiluted or diluted sera and ground suspensions of tissues obtained from patients by grattage were mixed and incubated in a water bath at 37°C. for 30 minutes in some experiments and 60 minutes in others. During incubation, the mixtures were agitated from time to time to insure uniform exposure of the tissues to the sera. After this period, the mixtures were inoculated in monkeys (M. rhesus) by swabbing the everted conjunctiva of one eye, and, in the opposite eye, by multiple pricking of the conjunctiva with a charged needle and injecting subconjunctivally 0.2 c.c. or more of the incubated reagents.

Experimental

In order to acquire immunological evidence for the presence of rickettsiae in trachoma, tests were made for the presence of agglutinins for Proteus bacillus, OX_{19} and OX_2, in sera from patients and animals. It seemed wise, moreover, to determine the converse of this—whether any antibodies capable of neutralizing the infectious agent of trachoma are demonstrable in antiproteus serum. The results of these tests will be given in brief.

Tests for agglutination. Agglutinations were done with sera of patients and animals. In the former case, serum was taken from 52 individuals, of whom 23 were Navajo Indians on the reservation in Arizona, and 29 were whites under observation in the Trachoma Hospital at Rolla, Missouri. In the different patients the disease varied in duration from a few months to 10 years, and the clinical manifestations were divided fairly evenly among the four types classified by Mac-

TABLE 1

OCCURRENCE OF THE WEIL-FELIX REACTION IN TRACHOMATOUS AND NORMAL INDIVIDUALS

Source of Sera	Number of Reacting Sera and Titer					Total Sera		
	1:5	1:10	1:20	1:40	1:80	Tested	With Agglutinins	Without Agglutinins
Trachoma								
(a) Indian	2	4	2	1	3	23	12	11
(b) White	1	1	1	2	1	29	6	23
Normal	1	3	2	1	1	15	8	7

Callan. For purposes of control, sera derived from 15 normal (that is, nontrachomatous) individuals were studied in parallel tests. With dilutions of sera running in successive, two-fold increments from 1:5 to 1:5,120, the agglutinations were conducted as described above. The results of these tests, summarized in table 1, reveal that 34 patients (11 Indians and 23 whites) possessed no agglutinins for either strain of Proteus bacillus even in the high concentration of serum of 1:5. On the other hand, 18 patients (12 Indians and 6 whites) yielded titers ranging from 1:5 to 1:80. Among the 15 normal individuals studied, 7 had no agglutinins and 8 manifested a scattering of titers similar to those observed among the reacting patients. It seems clear, therefore, that the frequency and distribution of agglutination does not vary materially between trachomatous and normal individuals, as other observers have also found.[8a] Whatever value, therefore, the Weil-Felix reaction may have in establishing the rickettsial nature of indeterminate infections, it apparently furnishes little, if any, assistance in the taxonomy of trachoma as a rickettsial disease. The interpretation of these results, however, will be considered later.

The lack of significant agglutinins in the sera studied precluded any incentive for further observation in patients. Consequently, agglutinations from then on were continued only with the sera of different animals. The results of these tests are given in table 2. It will be seen that sera were derived from eight monkeys (M. rhesus) with active, experimental

TABLE 2

AGGLUTINATION OF PROTEUS BACILLUS, OX_{19}, IN DIFFERENT SERA

Sera		Dilution of Sera					
Number	Source	5	20	80	320	1280	5120
8	*M. rhesus* Experimental trachoma	−	−	−	−	−	−
4	*Rabbit* Testicular passage, trachomatous tissues	−	−	−	−	−	−
2	*M. rhesus* Artificially immunized with trachomatous tissues	−	−	−	−	−	−
2	*Rabbit* Artificially immunized with trachomatous tissues	−	−	−	−	−	−
2	*Rabbit* Immunized with OX_{19}	++++	++++	++++	++++	++±	+

trachoma dating from one to six months; from four rabbits bled immediately after removal of testicles utilized for passage of the infectious agent;[11] and from two monkeys and two rabbits immunized artificially with trachomatous tissues as described earlier. In each instance, there was a complete absence of agglutination when strain OX_{19} was used as antigen. In sharp contrast, it is to be noted, were the results observed in tests with the antisera prepared by the immunization of two rabbits with Proteus bacillus OX_{19}. With patients or animals. In any case, moreover, the presence or absence of this reaction in trachoma can reveal only one phase of the immunological process. If, therefore, the data are to be inclusive, it becomes necessary to supplement the observations on the Weil-Felix reaction with a study of the protective capacity of a serum containing a high titer of agglutinins for the organism in question. Accordingly, suspensions of tissues from patients were "fixed" in the manner already indicated.

TABLE 3

PROTECTIVE CAPACITY OF ANTIPROTEUS (OX_{19}) RABBIT SERA IN EXPERIMENTAL TRACHOMA

Pooled Grattage Material from	Dilution of Sera	Period of Incubation	Infectivity in Monkeys of	
			Control Material	Test Material
4 patients	1:1	1 hour	4 inoculated 2 infected	4 inoculated 4 infected
2 patients	1:1	1 hour	4 inoculated 3 infected	3 inoculated 1 infected
2 patients	1:1	½ hour	2 inoculated 1 infected	2 inoculated 2 infected

this antiserum, agglutination of the homologous strain was obtained to an ultimate dilution of 1:5,120. In view of these observations further experiments with animal sera appeared to be superfluous. The inference, however, is obvious, that the antigen of the infectious agent of trachoma does not stimulate antibodies reactive with strain OX_{19}, even after prolonged artificial immunization.

Tests for neutralization of the infectious agent. That the infectious agent of trachoma is in the final analysis an ineffectual antigen has been long recognized from purely clinical experience. A recent communication from this laboratory[10] has now reinforced this belief with experimental evidence as well. For this reason, therefore, it should not be surprising that agglutinins for Proteus bacillus were not demonstrated in the sera of

In order to illustrate both the operation and the results of tests performed on neutralization or protection, the pertinent data are tabulated *in toto* (table 3). While several experiments were undertaken, only the three recorded were satisfactory since the original material used in the other tests was not infectious for monkeys. Equal quantities of trachomatous tissues and serum, and for control, similar mixtures of tissues and veal infusion broth, were incubated in two instances for 60 minutes, and in one instance for 30 minutes. Immediately following the period of incubation, the different mixtures were inoculated as shown in the protocol. The results of the experiment are so definite as to require little summarization. It is readily observed that in each test, anti-OX_{19} serum of high agglutinin titer (1:5,120) was incapable

of neutralizing the active agent of trachoma. The minor discrepancies between animals inoculated with tissues and serum and tissue and broth are obviously referable to variations in individual susceptibility. It seems, therefore, that mutually reactive antibodies have not been demonstrated in this study either in human or animal trachomatous sera or in anti-proteus serum.

Discussion

The evidence that has been offered to support the rickettsial origin of trachoma may be divided conveniently into three varieties—morphological, tissue cultural, and serological. It is true that Cuénod and Nataf[5] reported successful implantation of the infectious agent in the body louse, from which they characterized the agent as rickettsial. This, however, may have been as much the result of adsorption as implantation, and the experiments by Trapesontzewa[12] and Ruata[13] are not in accord with their observation.

The evidence derived from morphological studies is difficult of appraisal, since the ultimate interpretation of the particles seen under the microscope becomes more a matter of opinion than fact. Thus, for example, there is not only discord among those who consider trachoma a rickettsial infection as to which structures are the actual rickettsiae,[4, 5] but there are also as many competent observers,[8b] equally positive that no structures resembling rickettsiae are present in trachomatous tissues. It is interesting in this connection that Prowazek and Nicolle, both distinguished investigators of trachoma and rickettsial disease, never considered rickettsiae in their publications on trachoma. It is our experience that occasionally nondescript bodies may be seen in stained preparations which might conceivably suggest rickettsiae, but they are always numerically few, undistinguishable, and never do they give the appearance usually associated with similar preparations from authentic rickettsial diseases (in this connection see Rapisarda[8b]).

The tissue cultural studies have been disappointing in a clarification of the problem since, in our opinion, the infectious agent has not been propagated. Poleff* alone at the present time reports success with techniques which have failed consistently in others' hands. However, until Poleff carries out successful inoculations in man, ape, or monkey with the tissue cultures he regards as growth of the infectious agent, his conclusions must rest upon unstable evidence, being as it is morphological.

Attempts to establish trachoma as a rickettsial disease by way of the Weil-Felix reaction are based on the presence of agglutinins for Proteus bacillus in typhus, Rocky Mountain spotted, and trench fevers, all admittedly rickettsial manifestations. The common reaction is apparently due to a similar antigen in the rickettsiae of these diseases and Proteus bacillus[14] but it does not necessarily follow that all rickettsiae possess immunologically related antigens. Those who regard the reaction positive in trachoma acknowledge themselves too great an irregularity in the reaction and neglect to consider appropriately the possibility of preëxisting or concurrent rickettsial or proteus infection, and that agglutinins may occur even in so-called normal individuals. In any case, if the reaction were genuine, it is reasonable to expect a degree at least, of reciprocal reactivity between the respective antisera. This possibility has been subjected to trial for the first time in this study, and, as already

* Successful cultivation was reported recently by Rotth[6] also. In a personal communication later to Dr. Phillips Thygeson, he disclaimed his observations.

stated, antiproteus serum does not cross react with the agent of trachoma, thereby indicating again a lack of antigenic relationship between the agent of trachoma and Proteus bacillus.

It would take this communication far afield to enter upon a general discussion of rickettsial diseases. This has already been done[1] and it was shown that trachoma manifests none of the attributes assigned to the rickettsial diseases recognized at the present time. In short, it is the writers' opinion that what evidence has been assembled to prove the rickettsial origin of trachoma is inadequate and accordingly creates a justifiable hesitancy in its acceptance.

Conclusions

1. The majority of sera from patients with trachoma tested in this study contained no agglutinins for Proteus bacillus, OX_{19} and OX_2.

2. Patients' sera which caused agglutination did so only within the range observed among normal individuals.

3. Sera from monkeys infected experimentally or from rabbits serving for testicular passage of the infectious agent were devoid of agglutinins.

4. Sera from animals at the end of repeated, intravenous injections of trachomatous tissues similarly did not react with Proteus bacillus.

5. Antiproteus serum of high titer did not neutralize the infectious agent of trachoma.

6. The results of this study do not confirm observations by other workers on the occurrence of the Weil-Felix reaction in trachoma.

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FUNCTIONAL TRAINING, AN AID IN THE SURGICAL CORRECTION OF STRABISMUS

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The surgical correction of strabismus without further aid will produce satisfactory anatomical and physiologic results in a very small percentage of cases.

The surgical correction of strabismus from the standpoint of the cosmetic result is successful in a very large percentage of cases, if properly and carefully performed and with use of the proper technique.

In a previous paper[1] directed towards the technique of the recession operation, it was pointed out in a casual way that muscle training is of great importance, before and following the surgical correction of strabismus. This paper is to emphasize this point and to give some details concerning the management of cases from the beginning until desired results are obtained, exemplified in case histories.

Surgeons who have had only a moderate amount of experience in the surgical correction of strabismus can remember cases in which perfect anatomical and most excellent functional results have been obtained from a simple uncontrolled tenotomy. They have also seen cases of simple tenotomy with very discouraging results, such as marked overcorrection or undercorrection.

Haphazard surgery of this sort should be discouraged and only the type of surgical technique used which has been proved to have merits beyond question. To employ the highest type of surgery is but to take a small step toward the production of a physiological result.

The patient's attitude toward surgical correction of strabismus. The patient's ideas concerning strabismus surgery are varied. If he is quite young his attitude is to ignore it altogether; but when he is old enough to realize his physical handicap, his first thought is to secure an anatomical correction for the cosmetic effect. No until he is apprised of the fact will he understand that the most difficult part of the job is to make him use the two eyes together; in other words, to secure a functional result as well as a cosmetic one.

Etiology. The cause of strabismus will not be discussed in detail here, but it is well to call to mind what takes place from a physiological standpoint. In some cases, the patient has diplopia in the first stage of strabismus. In other cases, no doubt, suppression of vision precedes strabismus and diplopia does not exist. A deficiency or a complete loss of the fusion faculty is the most important factor in the etiology of strabismus.[2]

Physiologic changes. In the young patient the condition of diplopia is soon overcome by the physiological cessation of recognized vision in the deviated eye, known as suppression of vision. A series of physiological changes now takes place. Along with suppression of vision, is the suppression of the physiologic urge for single binocular vision, suppression of the act of fusion, and cessation of the further development of the visual neuronic paths and visual centers. This, in turn, soon lessens the functional powers of the visual neuronic apparatus and results in a diminished visual acuity which becomes a permanent visual defect. There is also

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developed a so-called false fovea, which is a physiological tolerance of an anatomical defect.

Management of strabismus. Ideally, the eye surgeon should have charge of all cases from their incipiency, but ignorance and procrastination on the part of the patients are at least two factors which block the initiation or progress of early treatment. A marked reduction in vision of the deviated eye is the result in a very high percentage of the cases.

The treatment of strabismus can be conveniently outlined in five stages;[3] namely, (1) refraction, (2) occlusion, (3) functional training, (4) surgery, and (5) postoperative training.

Early operation in young individuals is not usually indicated but rather discouraged, there are at least two reasons for this: first, much can be done by refraction, occlusion, and training; second, better operative results can be obtained after the patient is old enough to endure surgery under local anesthesia, and to give some coöperation to the surgeon.

The surgeon should understand orthoptics so that he can coöperate to the best advantage with the orthoptic technician. This will insure a careful study of the patient's condition by both. If the orthoptic technician is not present during the examination, he should be given all the details of the surgeon's findings and plan of treatment to guide him in his own examination and treatment.

What can one expect from nonsurgical treatment of strabismus? Penman[4] said, "There is no doubt that operation is needed less often when orthoptic training is employed."

Guibor[5] gives the number of corrections with refraction, atropine, and occlusion as about 30 percent and states that in about 50 percent of cases strabismus can be corrected by those means plus orthoptic training. He adds that the percentage of corrections is greater in squint below 15 degrees.

Emerson[6] believes that about 75 percent of cases of monolateral convergent concomitant squint with amblyopia ex anopsia are suitable for orthoptic training.

Daily[7] states that lower degrees of strabismus may be treated by orthoptic training alone while the higher degrees of strabismus should be treated with orthoptic training plus surgery.

Post[8] predicts that apparently orthoptic training will add an additional 20 or 30 percent to the good results obtainable merely by suitable glasses.

These citations confirm my own conclusions from personal experience and observation in the use of orthoptic training. The age of the patient has an influence on the results obtainable. Before the sixth year better results can be obtained than after the sixth year, and after the eighth year there is a marked drop in the percentage of successes.

It is not within the scope of this paper to give detailed instruction concerning the technique of orthoptic training except to say that the modern instruments for orthoptic training have a great advantage over the older methods, but should not entirely replace them.

The following case reports will serve to illustrate the benefit of nonsurgical treatment for strabismus.

Case Reports

Four groups are presented:

Group A: Cases in which the squint was corrected by the wearing of lenses prescribed as the result of cyclopegic refraction and in which training was not necessary.

Group B. Cases in which lenses were prescribed and worn but the squint was corrected only after orthoptic training.

Group C. Cases in which the wearing of proper lenses and subsequent training

did not correct the existing strabismus, and surgical correction was required.

Group D: Cases in which operation was not preceded by training, but orthoptic training was given postoperatively.

Group A consisted of 31 cases.

Group B, 12 cases, was classified as follows: Nine hyperopic cases of monocular convergent concomitant strabismus, two cases of myopic monocular divergent concomitant strabismus, and one of hyperopic alternating strabismus, successfully corrected by the method of refraction, occlusion, and functional training.

Suppression of vision was present in all cases and cessation of fusion was present in all except two of the convergent strabismus cases, in each of which the patient could fuse at certain times only.

In all except four cases there was defective vision in the deviated eye and normal vision in the fixating eye. The four exceptions were, one case of concomitant convergent strabismus, one of alternating strabismus, one of divergent strabismus (in all three of these there was normal vision in both eyes), and a case of divergent strabismus with defective vision in both eyes.

The number of orthoptic treatments varied from 10 to 68, the case of alternating strabismus requiring the most treatment, and the case of divergent strabismus with normal vision in both eyes requiring the least number of treatments.

In group C, there were 38 cases of strabismus treated by orthoptic methods. These did not correct the strabismus and the patients were operated on after from 2 to 13 months of training. Of these 38 cases, 6 needed no postoperative training, 5 did not come in for further training, and 27 received postoperative training. Six of these patients developed no fusion; 4 developed grade-1 fusion; 3 developed grade-2 fusion; and 14 developed grade-3 fusion. The number of postoperative treatments ranged from 12 to 52.

Group D comprised 53 patients with strabismus who were operated on, but who did not receive orthoptic training previous to the surgical procedure. They were mostly young adults who were seeking cosmetic results only. Thirty-one of them were persuaded to take postoperative training. Among these were 24 cases of convergent squint, 3 of divergent squint, and 4 of alternating strabismus. In six no fusion nor even the slightest signs of fusion could be developed. Five patients developed grade-1 fusion, 5 developed grade-2 fusion, and 15 developed grade-3 fusion, the number of treatments ranging from 15 to 57.

From the results thus far obtained, I would emphasize strongly the importance of preoperative orthoptic training. In some instances the eyes can be straightened and in others the degree of squint reduced with orthoptic training. But, in any instance, the important problem is to teach the patient basic eye functions, such as accurate monocular fixations, good monocular rotations, good accommodative responses, fusion, and the breaking down of suppression; also the functional training of all centers pertaining to vision and to the control of visual organs. Then when an operation is performed, and the patient has a straight pair of eyes, he will have some idea of how to use them. The small adjustments that may have to be made can be taken care of in some cases by the patient himself through proper use in those first few weeks after the operation, before more detrimental ocular habits are formed. Without training the old bad habits are too apt to persist and after an operation are difficult to break,

and the case takes longer to reach a satisfactory conclusion. If the patient has had the preliminary orthoptic training and the eyes have been straightened by surgery, the postoperative exercises can be directed toward the building of ductions and stereopsis.

Early operation is not always indicated. This is especially true in cases in which the patient lends himself to good coöperation; for in these cases it is assured that the patient will receive training as often and as regularly as is necessary. Only in those cases in which regularity cannot be kept in the training, due to the lack of understanding or indifference on the part of the parents or for other causes, it is best to operate earlier. Even then the surgeon cannot depend upon orthoptic training as an aid to surgery, and for the same reason.

C. S. Smith[9] thinks that surgery is indicated in every case of strabismus that is not cured after six months to one year of energetic nonsurgical treatment.

Martin[10] stresses the importance of training the public as to the value of surgical correction of strabismus, and, I would add, the training of the medical profession as well.

Surgery should be employed with scarcely an exception in all cases in which well-selected and well-applied nonsurgical treatment has failed to bring the desired results.

The time limit for the application of the training methods is one that is open to dispute. Experience has led me to believe that the period of training may well be lengthened in the young until they have reached the age at which they can be operated on under local anesthesia, which, of course, depends much upon the disposition, training, and the coöperative ability of the patient.

Should all operative cases receive orthoptic training, both before and after the surgical correction of strabismus? The question of orthoptic training before surgery is answered only by the case in question.

In all younger patients, if it is possible, orthoptic training should be used before an operation is attempted. In older patients with fair vision in the crossed eye and low degrees of squint orthoptic training will be an advantage, for while it cannot be expected that such training will correct the squint in a large percentage of cases, it will strengthen the weak muscles and develop their controlling center. Physiologically, it will strengthen the urge for fusion and develop the normal retinal correspondence—all of which will be a distinct advantage toward securing the desired result following surgical correction.

As to using orthoptic training following operation this depends on many factors; first, the operation itself. I am convinced that the less trauma a muscle receives, the better its spontaneous response and the better the results from orthoptic training. The surgeon should ever keep this in mind while planning and performing the operation. A few, especially of the younger patients will have sufficient amplitude of fusion to stimulate binocular single vision, which may automatically produce normal function. In those few cases, therefore, training will not be necessary.

In most cases, however, orthoptic training should be given after operation. In a paper entitled "Surgical results in 223 cases of heterotropia," Berens[11] found that "25 percent of 126 patients, with heterotropia had false projection, and following orthoptic training the number was reduced to 10 percent."

Bressler[12] in reporting 218 cases of strabismus corrected by surgery found that in one group which had not received orthoptic training, either before or after

surgery, there were very few cases of spontaneous fusion while in another group which had received orthoptic training both before and after surgery, fusion resulted in 81 percent.

Summary

Of the 134 cases here reported 81 were treated by orthoptic methods, of which 43 were corrected by refraction, occlusion, and orthoptic training. Thirty-eight received preoperative training and 27 were given both preoperative and postoperative training. Thirty-one received postoperative training only and 22 operative cases received no training at all.

Conclusions

About 50 percent of all cases of strabismus can be corrected by refraction, occlusion, atropine, and orthoptic training.

Early operation is not necessary in a large percentage of cases, if training can be used regularly.

It is of great importance to have the coöperation of the parents and teachers.

Most patients, especially in the younger groups, should have orthoptic training before operation.

All with but few exceptions, should have postoperative orthoptic training.

Functional training has its effect upon the visual centers and the centers controlling the movement and behavior of the visual organs.

Early consideration of squint is of paramount importance, as surgery may be reduced to a minimum by starting those cases as early in life as possible.

The author thanks Miss Alice Burt and Miss Vivian Ilg of the orthoptic department for their valuable service in the training of these patients.

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MALIGNANT MELANOMA OF THE CHOROID AND VON RECKLINGHAUSEN'S DISEASE

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Malignant melanoma has long been known as melanosarcoma. While this tumor is well known to ophthalmologists, there are many features about it that need clarification. It is primary in the eye, the skin, and exceptionally in other organs. The association of malignant melanoma and von Recklinghausen's neurofibromatosis in the same individual has been rarely noted. Only three other cases with this combination have been found in the literature.

Von Recklinghausen's disease[1] is characterized by tumor formation on the roots and branches of the cerebrospinal and sympathetic nerves. The tumors often appear in the cutaneous nerves with the formation of characteristic skin nodules. Some cases develop plexiform neuromas, others elephantiasis. Localized growths may occur in the brain and spinal cord. The common tumor of the cerebellopontine angle is a neurofibroma of the acoustic nerve.

Melanomas and von Recklinghausen's disease are characterized by a number of features that are so similar that they indicate an association of these two diseases. Nevi probably belong to this group.

Malignant melanoma is rare. In studying its incidence (Agatston and Gartner,[2] 1939) we found that of one million of the white population less than two died each year from melanoma. Von Recklinghausen's neurofibromatosis is also rare. Although there are no definite figures available, one may judge from Beck's[3] study (1938) of 595 autopsies on tumors of the nervous system, 7 cases (1.17 percent) were of neurofibromatosis. These two diseases are so rare that their occurrence in the same individual could hardly be a coincidence.

CASE REPORT

R. S., a white woman of 54 years, called at the clinic of Dr. Bernard Samuels at New York Hospital. She complained of a discharge from the socket of her left eye.

She was a nurse at Montefiore Hospital 10 years ago, when she noticed blurring of the vision of her left eye. A detached retina was found. Transillumination was positive. Malignant melanoma of the choroid was diagnosed, and the eye enucleated by Dr. S. A. Agatston.

For many years the patient had noticed the slow growth of many small nodules in her skin. These were painless and only annoyed her by their appearance.

Twelve years ago, when her age was

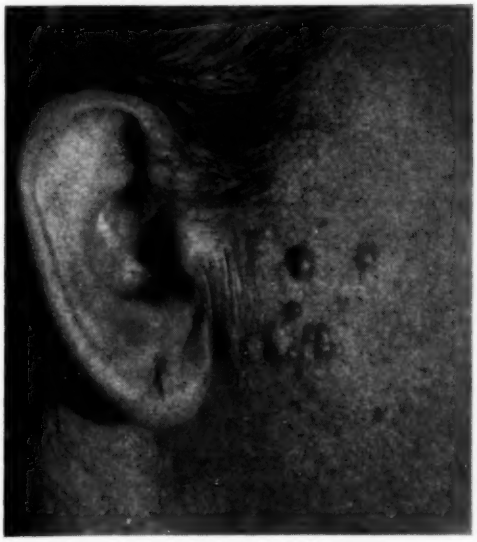

Fig. 1 (Gartner). Von Recklinghausen's disease.

Fig. 2 (Gartner). Malignant melanoma of the choroid (low power).

42 years, a hysterectomy was performed. Pathological diagnosis was multiple fibroids of the uterus. There were many subserous, intramuscular, and submucous fibroids.

Physical Examination

Skin: There were many nodules in the skin. These were especially numerous in the right cheek, the forearms, chest, and back. They varied in size from 2 to 10 mm. Some were pedunculated, most were attached to the skin by a broad base. The color in general was that of the neighboring skin. A few were darker and some had the typical café-au-lait pigmentation. The nodules were soft and gelatinous to touch. A few of the larger ones felt like "a bag of worms." None was tender. There were many pigmented nevi in the skin (fig. 1).

Head: Normal in shape.

Eyes: The right eye was normal. The pupil reacted promptly to light and accommodation. The fundus was negative. Vision was 20/20.

The left eye had been enucleated. The patient wore a well-fitting prosthesis which moved with the other eye. The edges of the glass eye were slightly roughened. The conjunctiva was congested. The socket was in good condition, with no sign of tumor formation nor pigmentation.

Ears: There was a slight discharge from both ears. The drums were perforated. Hearing was impaired in both ears, due to middle-ear disease.

Nose and throat were negative. The glands of neck were not enlarged. The heart and lungs were negative. The abdomen had a low mid-line, post-operative scar. Liver and spleen were not palpable. The extremities were negative. Neurological examination disclosed no abnormalities.

The urine was negative for pathology; the melanin test, negative; and the Wassermann test, negative.

A biopsy of one of the skin nodules from the left forearm was submitted to Dr. N. C. Foot at New York Hospital. He reported it a typical neurofibroma.

Some of the sections of the enucleated eye were on file at Montefiore Hospital and available for study. Dr. Bernard Samuels examined them and confirmed the diagnosis of a typical malignant melanoma (see figs. 2 and 3). The growth was confined to the choroid. There was a low

detachment of the retina over it. The tumor was full of dark-brown pigment, intracellular and extracellular. The cells were closely packed and mainly spindle shaped.

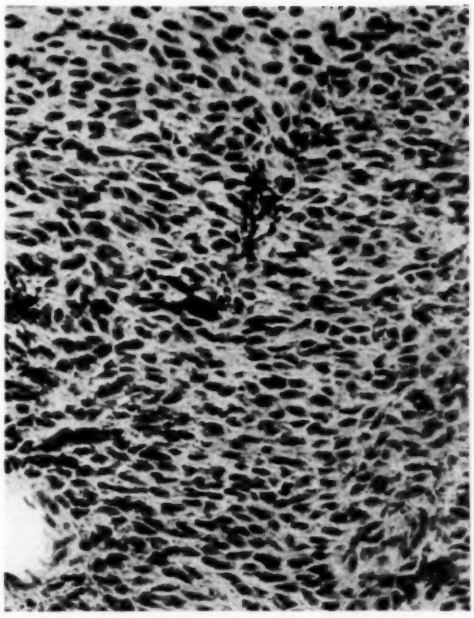

Fig. 3 (Gartner). Malignant melanoma of the choroid (× 240).

Association of Malignant Melanoma and von Recklinghausen's Disease

Melanin. Malignant melanomas derive their name from the abundance of melanin they contain. The pigment is not an essential part of the cells, for some of these tumors contain no melanin. They are then called leukosarcomas. Careful search of these growths usually discloses a small amount of melanin, rather than a complete absence. Nevi are also characterized by melanin deposits. This is of interest, as it has been repeatedly demonstrated that some nevi develop into malignant melanomas. In von Recklinghausen's disease, the skin overlying the neurofibromas as well as other cutaneous areas are often pigmented. Characteristic is the café-au-lait pigmentation. This pigment is melanin.

Dopa reaction. Bloch (1917)[4] made extensive studies of the dopa reaction, which he found positive only in ectodermal tissue. The dopa-positive cells contain a specific oxidase which changes the colorless melanogen in the blood to the pigmented melanin. Bloch found cells in malignant melanoma and nevi to be dopa-positive. Laidlaw (1932)[5] found dopa-positive melanoblasts in cases of von Recklinghausen's disease.

Origin of malignant melanoma and von Recklinghausen's disease. Malignant melanoma has long been considered mesodermal and was therefore called melanosarcoma. Neurofibroma was thought to arise from the connective tissue of the endoneurium or perineurium. The ideas on this subject have changed, so that now it is thought that both these tumors arise from the neuroectoderm. Von Recklinghausen[1] (1882) described neurofibroma as mesodermal. Soldan (1889)[6] claimed nevi were a phase of neurofibroma. He was the first to demonstrate an intimate connection between a nevus and the sensory nerves of the skin. Verocay (1910)[7] described the origin of neurofibroma from Schwann-sheath cells. The Schwann layer of syncitium envelopes the peripheral nerves. It has been called the peripheral neuroglia. This has been proved to be definitely ectodermal in origin.

Masson (1932)[8] proved neurofibromas are neuroectodermal in origin and demonstrated their derivation from the Schwann layers of syncitial cells that surround the nerves. He asserted that nevi and melanomas are derived from nerve end-organs. He found a relationship to neurofibromatosis and showed many features of nevus formation in the pigmented spots of von Recklinghausen's disease. He observed miniature neurofibromas in connection with pigmented nevi.

The Wagner-Meissner corpuscles are the sensory-nerve endings normally found in the digital pulp of the skin. With tumor formation, sensory nerves in other parts of the body have a tendency to form structures resembling these corpuscles. Masson (1932)[8] has shown the development of similar Wagner-Meissner corpuscles in nevi, malignant melanoma, and neurofibromatosis.

The work of Masson has been confirmed and championed by others. Most noteworthy are the studies by Ewing (1922, 1928)[9] and a group at Memorial Hospital including Quick and Cutler (1927),[10] Stewart and Copeland (1931),[11] and Adair (1935)[12]; at New York Hospital by Foot (1931–1936),[13] and at Johns Hopkins by Geschickter (1935),[14]

Simon and Levy (1923)[15] and Bertrand and Bernard (1930)[16] reported cases of malignancies in von Recklinghausen's disease which they called Schwannomas —a growth of Schwann-sheath cells about the nerves. Abbot and Jehiel (1932)[17] reported a similar case and called the malignant tumor a peripheral glioma. Ewing, Quick, and Cutler, and Stewart and Copeland believe the malignancies of von Recklinghausen's disease are neurogenic sarcomas.

Laidlaw and Murray (1933)[18] found an anthropological basis for nevi. They demonstrated the similarity of nevi to the pigmented sensory-nerve terminals of reptiles and amphibia. They suggested that nevi were a step in the evolution of tactile corpuscles.

Malignancies in von Recklinghausen's disease. Von Recklinghausen's disease has a great tendency to develop malignancies. Garré (1882)[19] reported 12 percent of his cases developed malignancies. Fischer (1927)[20] collected 466 cases of von Recklinghausen's disease and found that 13 percent developed malignancies. Many began after partial removal or injury of a neurofibroma. Hosoi (1931)[21] found that 22 percent of 65 cases that had become malignant had metastases.

Of Fischer's 466 cases, 299 were in men, 167 in women; almost twice as many in men as in women. The same proportion was found in the group which developed sarcoma. In a previous study (Agatston and Gartner, 1939[2]) 755 cases of malignant melanoma were collected, of which 421 were in males, 304 in females; also showing an actual preponderance of males.

Hosoi's 65 patients with malignancies in neurofibromatosis developed their malignant degeneration between the ages of 16 and 70 years; most were between 30 and 60. In the previous study referred to, a similar age grouping was found for melanoma. Of 550 patients, the greatest number were between 40 and 60 years of age.

Neurofibroma in the eyeball. The occurrence of neurofibroma of the eyeball has been noted a number of times. Interesting reports were made by Callender and Thigpen (1930),[22] Copeland, Craver, and Reese (1934),[23] and by Freeman (1934).[24] Wheeler (1937)[25] reported neurofibromatosis of the eye, mainly involving the choroid and ciliary body. He pointed out that in some portions of the neurofibroma, structures resembling Meissner corpuscles were found, similar to those previously described in nevi and malignant melanomas. In the eye, neurofibromas have been found in all the tissues except the lens, but mainly in the uveal tract. The distribution is similar to that in malignant melanoma, most occur in the choroid, fewer in the ciliary body and iris.

Schubert (1925)[26] suggested that some cases of malignant melanoma may be due to degeneration of a neurofibroma. Stough (1937)[27] reported a case of solitary intraocular neurofibroma which arose

from a ciliary nerve. He suggested the probability that some reported cases of malignant melanoma, particularly the leukosarcoma, were really cases of neurofibroma. He described many similarities in the appearance and arrangement of the cells.

Malignant melanoma of the choroid. Dvorak-Theobald (1937)[28] made a study of melanoma of the choroid and its relationship to the ciliary nerves. She presented seven cases in which every section of the tumor was studied; with the aid of special silver staining the ciliary nerves were traced into the tumors. Her observations led her to believe that the melanomas originated from the Schwann-sheath syncitium of the nerves. Dvorak-Theobald stated that the most common site for uveal tumor is to either side of the optic foramen in the horizontal meridian; that is, along the path of the long posterior ciliary nerves. Anteriorly, it is in any quadrant.

Nitsch (1929)[29] pointed out that sarcomas mainly originate in the outer portion of the choroid, where most of the nerves lie.

Cases of malignant melanoma and von Recklinghausen's disease. Few cases have been reported with the association that our case presents–of malignant melanoma and neurofibromatosis. Berblinger (1915)[30] reported a case of multiple malignant melanoma of the brain and neurofibromatosis, with a complete autopsy study. Björneboe (1934)[31] reported a case of malignant melanoma of the brain, neurofibromatosis, and extensive nevi, with an autopsy study. Bair and Love (1937)[32] reported from the Mayo Clinic a case with malignant melanoma of the choroid and acoustic-nerve neurofibroma. Both were removed and proved pathologically. None of these authors offered an explanation of this association.

Summary

The occurrence of malignant melanoma of the choroid and von Recklinghausen's disease in the same individual is reported.

Evidence is submitted for grouping these diseases as neurogenic tumors in a related etiology.

The following are considered to be features of their association: 1. Melanin is found in both. 2. Dopa reaction is positive for both. 3. Both originate from neuroectoderm. Malignant melanoma of the choroid has been traced anatomically from the ciliary nerves. Neurofibromas are intimately associated with the nerves from which they arise. Both have been traced from the Schwann layer of syncitial cells surrounding the nerves. 4. Neurofibromas have been found in the eye, mainly in the uveal tract. They are located in the same regions as malignant melanomas. 5. Both show a tendency to form structures resembling Wagner-Meissner tactile corpuscles. 6. In von Recklinghausen's disease, 13-percent of the cases undergo malignant degeneration. It has been suggested that some cases of malignant melanoma develop from neurofibroma. It has also been suggested that some cases diagnosed as malignant melanoma, particularly the leukosarcoma, are really neurofibroma. 7. Three previous cases have been reported in the literature, showing the association of malignant melanoma and von Recklinghausen's disease in the same individual.

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NOTES, CASES, INSTRUMENTS

A NEW ABSORBENT SPONGE FOR EYE SURGERY*

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In the early days and before asepsis was known in surgery, sea sponges were used to pick up the blood and body fluids during operative procedures; but after the germ theory had been proved a fact sea sponges fell into disuse, because they could not be properly sterilized by any known method.

In going through the literature, it was found that for several years back, very little has been written concerning absorbent sponges for eye surgery, and it is presumed that this is true also in other branches of surgery.

In the older medical literature, the sea sponge is described as being the most ideal material for surgical use. In Beard's[1] book on ophthalmic surgery he states, "The property most essential in a sponge is great absorptivity." In another book[2] on surgery it is stated that "Sea sponges have a greater absorbing power and are more elastic and yielding than gauze sponges" . . . "Sea sponges[3] are no longer used in surgery, but they were for a long time considered an essential part of surgical technique." . . . "Gauze[3] pads are not so soft and elegant as marine sponges, but the former can be made sterile by steam, while the latter do not bear heat well in any form, and all methods of sterilizing them have proved disappointing."

This apparently establishes (by those who used them) the fact that highly absorbent material like sea sponges is more ideal than cotton or gauze for surgical sponges.

It appears that the surgeons have long since dismissed the subject of sponges for surgery and have for the most part settled down to the use of the conventional type of gauze pads and cotton pledgets. This, no doubt, is due to the fact that no material has yet been found that is better than gauze pads and cotton pledgets, or that will in any way approach sea sponge for surgical use.

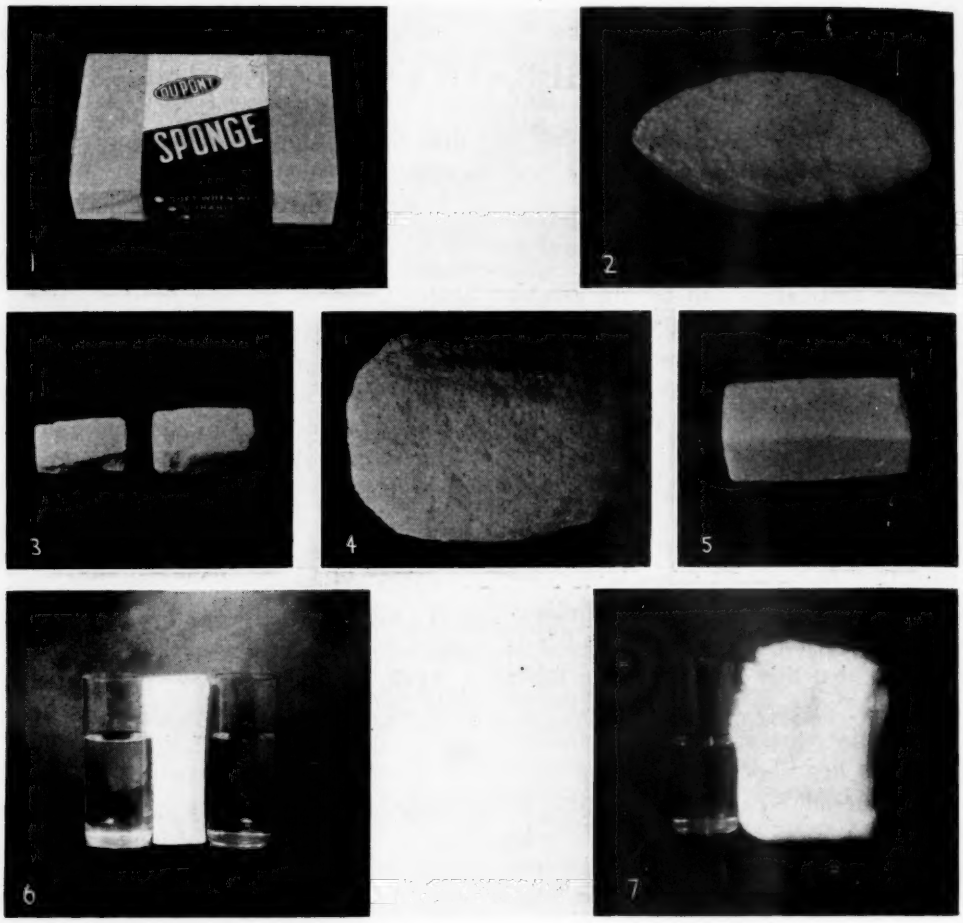
The tightly rolled moist cotton pledget has been rather generally accepted as the best type of sponge available for eye surgery; some eye surgeons use gauze pads especially for lid surgery.

Again Dr. Beard states[4] "Dry cotton is most objectionable in eye surgery." It is objectionable because of the loose fibers which may adhere to the surgical instruments, also because dry cotton is very slow to absorb moisture. Liquids are taken up by cotton much more rapidly if its fibers are moist, but it is a fact that a given amount of dry cotton will take more liquid than the same amount of wet cotton.

Cotton is used for the manufacture of eye sponges, perhaps for the simple reason that it is easy to obtain, that it can be safely sterilized, is rather inexpensive, and, especially, that no better material has been brought forward to take its place.

I have, for quite a few years, been searching for some fabric from which a more ideal surgical sponge could be manufactured, but every experiment that I tried proved worse or no better than the conventional cotton sponge. Lamb's wool, a mixture of lamb's wool and cotton, also cotton and gauze were tried; and experi-

* From the Ophthalmic Department of the Chicago Eye, Ear, Nose, and Throat Hospital.



Figs. 1 to 7 (Nugent). 1, cellulose sponge in original package, size 4⅝ by 6½ by 2¼ inches; 2, eye sponge cut to a convenient shape for intraocular surgery, 1¾ inches long by one-half inch thick; 3, sponges for lid and lacrimal surgery—identical in size when dry—the one on the right is moist, showing increased size, the dry sponge is 1⅛ by one-half by one-half inch in size; 4, convenient shape for tonsil surgery, 1½ by 1½ by 1 inch in size; 5, shaped for general surgery; 6, a sponge, 6¼ by 3⅞ by 2 inches in size, from which 600 c.c. of water has been expressed; 7, cotton of the same weight as the sponge in figure 6 held only 200 c.c. of water.

ments with an absorbent paper pulp were unsuccessful.

A few months ago, in a hardware store, I discovered an artificial sponge for commercial use, manufactured by the Du Pont Company and called cellulose sponge. I bought it and took it home with me for experimental purposes. It was found to be made of a material that had the highest absorbent qualities of any that I had ever seen. The pores in the sponge, however, were too large to make it a practical eye sponge. I got in touch with the manufacturers and found that they were just beginning to manufacture a sponge with smaller pores, more compact, and of a finer texture.

This type of sponge proved to be ideal. It is exceedingly soft and pliable. It will take up more than 20 times its weight in water, can be boiled again and again and still retain its softness, and, when fash-

ioned in shapes most convenient, proves to be a most superior sponge for surgical use.

We have fashioned sponges of this material for eye, tonsil, and mastoid surgery; also larger sponges for general surgery which have proved ideal.

These sponges are sterilized by boiling or in the autoclave. They can be washed and sterilized and used repeatedly. I have a set of sponges that I have used about 80 times and they still retain the same softness and high absorbent qualities that they had when first used.

Advantages

I do not know, nor can I guess, how many times the sponges can be used.

If the sponges become darkened or discolored from repeated use, they can be brought back to their original color by boiling in a weak solution of baking soda, two level tablespoonfuls to each quart of water; or by rinsing them in a solution of two tablespoonfuls of 3-percent peroxide solution in each pint of warm water (about 110° to 115° F.); or by placing them in a solution of Clorox, one tablespoonful to one pint of water, for one-half hour, after which time sponges will be thoroughly bleached, then boiling them for 20 minutes to remove all odor of Clorox, rinsing in cold water, and letting them dry.

Cellulose sponges come in a bricklike shape as large as 4⅝ by 6½ by 2½ inches, and the surgeon can shape them to any configuration and size he desires. It is a very simple procedure: Simply wet the cellulose sponge, squeeze the excess water out, then cut with scissors and shape to the desired form.

The accompanying photos show a few shapes suggested for special and general surgery.

231 West Washington Street.

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[1] Beard, C. H. Ophthalmic surgery. Ed. 2, Philadelphia, P. Blakiston's Son & Co., 1914, p. 31.
[2] Bryant and Buck. American practice of surgery. New York, Wm. Wood, 1908, v. 4, p. 138.
[3] ———. American practice of surgery. New York, Wm. Wood, 1906, v. 1, p. 720.
[4] Beard, C. H. Ophthalmic surgery. Ed. 2, Philadelphia, P. Blakiston's Son & Co., 1914, p. 35.

A PRISM SCALE*

Conrad Berens, M.D.
New York

A prism scale is useful in determining the optic center of correcting lenses in addition to determining the amount of prism in the patient's lenses and verifying the prisms furnished in ordinary trial cases and boxes of prisms.

The scale described in a previous communication[1] permitted the study of 50Δ prisms and was particularly useful in checking the accuracy of numbering. It was found to be somewhat less useful in measuring the amount of vertical prism in lenses.

The prism scale to be described (fig. 1A) has been designed primarily for the study of the amount of prism in the lenses the ophthalmologist prescribes. This scale** does not permit the study of the stronger prisms that are used especially in examining for motor anomalies with the screen test.

* Presented before the Section on Ophthalmology, American Medical Association, June 13-15, 1938. Aided by the Ophthalmological Foundation, Inc.

** Made by E. B. Meyrowitz Company, New York.

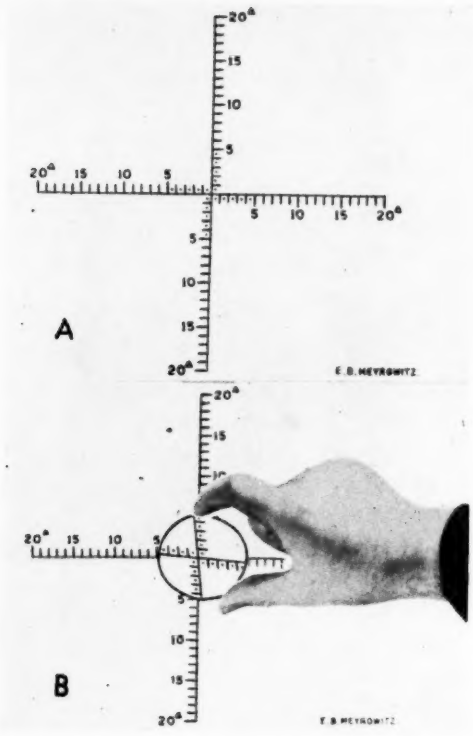

Fig. 1 (Berens). Prism scale. A, the anterior surface of the prism scale; B, determination of center of lens in astigmatic axis.

The former scale was made of solid bakelite, but this scale is made of black bakelite superimposed upon white bakelite attached to a thin board. When the black surface is removed the white markings are permanent and the entire scale is practically indestructible.

The bakelite board is 32 cm. by 21 cm., with a 50-cm. cord attached to the center of the scale where the vertical red line and the horizontal green line cross. The scale is marked in one-half prism diopters up to 5 and in 1$^{\Delta}$ markings up to 20$^{\Delta}$.

The scale may be used to measure prisms placed with their bases vertical or horizontal.

The observer should be sure that the eye he uses is opposite the center of the scale and level with it. The scale should be well illuminated, and the lens to be tested should be placed against the metal cylinder at the observer's end of the cord (fig. 1B).

The geometric center of the lens should be marked with a china marking pencil, and the amount of displacement of the vertical red line on the horizontal scale indicates the amount of prism, base in or base out, in the lens. If the green line is displaced in the vertical plane the amount of displacement of the green line against the red line indicates the amount of prism in the lens, base up or base down.

35 East Seventieth Street.

Reference

[1] Berens, C. Prism scale for use at 50 centimeters. Amer. Jour. Ophth., 1938, v. 22, p. 542

SOCIETY PROCEEDINGS

Edited by Dr. H. Rommel Hildreth

COLORADO OPHTHALMOLOGICAL SOCIETY

January 28, 1939

Dr. Melville Black, *presiding*

Symposium on sulfanilamide

Dr. Richard Whitehead, professor of pharmacology at the University of Colorado, presented a general pharmacological discussion of sulfanilamide and related drugs. The antistreptococcal action of azo dyes was first recognized in 1909. At this time Horlein found that one of the textile dyes had a limited therapeutic action against mouse septicemia caused by hemolytic streptococci. Further studies by Domagk resulted in the development of prontosil in 1932. Later a more soluble and less toxic compound was developed. This was introduced under various trade names as prontosil soluble or neoprontosil. Trefouel, Nitti and Bovet studied a long series of compounds and demonstrated that the chemotherapeutic action was due to sulfanilamide derived from prontosil in the organism. Sulfanilamide was first synthesized by Gelmo in 1908. (Trade names 1162F, Prontylin, Prontosil Album, Stramid, Sulfamidyl, Sulfonamide-P, Streptocide, Colsulamyde, Despeptyl, and so forth.) More than 200 related compounds have since been studied for their chemotherapeutic effect.

Sulfanilamide and related drugs have been shown to have curative effects in erysipelas, miscellaneous oral infections, puerperal infections, meningitis, cystitis, peritonitis, and in other diseases due to hemolytic streptococci. Encouraging results have also been obtained in gonorrhea, meningococcic meningitis, gas gangrene, pneumonia, and so forth.

The mode of action of sulfanilamide is not definitely known. Apparently it does not act primarily by the neutralization of bacterial toxins. Recently Locke, Main, and Mellon have advanced the following hypothesis: "The growing bacterial cell has the power to convert sulfanilamide into a derivative which is a highly active anticatalase. This results in the accumulation of anticatalase in the immediate vicinity of the cell. The streptococcus and related organisms being active producers of hydrogen are able to grow only so long as the peroxide concentration can be kept below a critical level by diffusion or destruction. Usually this is accomplished by catalase from the blood and tissues. In the presence of anticatalase, however, the catalase is inactivated in a zone adjacent to the cell, with the resultant accumulation of hydrogen peroxide to toxic levels."

Sulfanilamide is absorbed readily from oral administrations. It appears in all the body fluids and penetrates into the tissue readily as the concentration in the tissues parallels the water content. Sulfanilamide and related drugs are excreted readily by the normal kidney but less rapidly by the damaged kidney. The peak level of sulfanilamide in the blood is reached 48 hours after oral administration and is largely excreted in the urine in 48 hours. A detailed discussion of toxity and dosage was also given.

Discussion. Dr. Maurice Marcove discussed the use of sulfanilamide in trachoma and quoted from the paper of Fred Loe: "Sulfanilamide treatment of trachoma" (Jour. Amer. Med. Assoc., 1938, v. 108, Oct. 8, p. 1371).

Dr. Marcove then presented the cases

of three women who had acquired trachoma 2½ years ago, presumably at a Russian bath. All three had pannus and corneal ulcers. All had been treated with silver nitrate and copper sulfate. Two eyes had been tarsectomized. These patients were given sulfanilamide, 15 grs. t.i.d., with equal amounts of soda for one week. The dose was then reduced to 10 grs. t.i.d. for four weeks. No further medication was given. Within the first few days the inflammatory symptoms entirely subsided. The follicles began to recede after the first week and were almost gone at the end of the five-weeks period. Two of the patients had reactions. One had a marked skin rash and the other had extreme malaise and weakness. In both of these cases the drug was discontinued for a week and then resumed without consequent reaction.

ORBITAL CELLULITIS TREATED WITH SULFANILAMIDE

DR. J. LEONARD SWIGERT reported on the case of Miss H. T., aged 17 years. This patient was first seen on November 26, 1937, and a diagnosis of orbital cellulitis was made. The ethmoid sinuses were drained mechanically, and typhoid vaccine was given intravenously. The condition continued to grow worse, so an incision was made into the orbit on December 4, 1937. A large amount of foul-smelling pus was obtained. Pneumococci and streptococci were found in cultures. Forty grains of sulfanilamide was given daily from December 6th to December 12th. Marked improvement immediately followed the use of the sulfanilamide. When last seen in May, 1938, there was no evidence of the previous difficulty and vision was 20/20 in each eye.

DR. J. LEONARD SWIGERT also reported on the case of Mr. M. H., aged 23 years, who was seen on May 28, 1938, on account of proptosis and pain in the eye of one weeks' duration. The orbit was opened surgically and a moderate amount of serosanguineous pus was obtained. Hemolytic staphylococci and streptococci were found in culture. The condition continued to become worse after the surgery, the patient having a fever of 105.5 degrees. On May 31, 1938, 90 grains of sulfanilamide was given, and this dosage was continued for three days. By this time there was marked local improvement and the temperature was normal. Thirty grains of the drug was administered daily until June 18, 1938, at which time there was no evidence of infection. The eye remained blind from optic atrophy and a symblepharon had formed. This symblepharon was later surgically corrected.

SULFANILAMIDE IN GONORRHEAL CONJUNCTIVITIS

DR. HARRY SHANKEL discussed the dramatic effects of sulfanilamide in cases of conjunctival gonorrhea and presented abstracts of papers by Henry Ware Newman (Texas State Journal of Med., Dec., 1937), Claude S. Perry (Ohio State Med. Jour., Feb., 1938), Fernandez and Fernandez (Amer. Jour. Ophth., July, 1938), and Thayer Willis (Yale Jour. Biol. and Med., Jan., 1938).

John C. Long,
Secretary.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

SECTION ON OPHTHALMOLOGY

January 20, 1939

DR. FRANK N. KNAPP, *president*

CHRONIC CONJUNCTIVITIS

DR. SANFORD R. GIFFORD, Chicago, said he wished to make a plea for a more exact diagnosis in this field. The field

of chronic conjunctivitis has developed into a sort of garbage heap to which we consign everything we do not know how to classify. In the first place, before we attempt to treat chronic conjunctivitis, it is most important to determine if the patient has conjunctivitis at all. He may have chronic conjunctivitis or only an irritation of the eyes due to a variety of causes. Hence it is important to determine the presence of conjunctivitis. People who have a mild form of epithelial dystrophy with slight redness of the conjunctiva do not have real conjunctivitis. There are other cases in which the symptoms of irritation are due to common causes of irritation, chemical or mechanical, or to eye strain. Patients in the early presbyopic stages complain of vague irritation of the eyes. The eyes get red and at times there is a little secretion, but symptoms are due to overstrain of eyes and of course are corrected by proper glasses. Then we have cases due to chemical irritants such as smoke. A good many patients in Chicago who are commuters have slight redness of the conjunctiva; they have no real thickening of the conjunctiva, practically no secretion, but the eyes are slightly red. If one commutes in a smoking car twice a day the eyes are exposed not only to one's own smoke but that of others, and sometimes they are relieved by taking away that factor. Then there is the smoke of other occupations and other causes which can be eliminated. Such patients recover if the external irritants are removed. They are made worse with the usual astringents. What they need if anything is something which will make them more comfortable until the symptoms subside by themselves—such as adrenalin with a local anesthetic. Dr. Gifford does not like to use cocaine for these cases, or butyn which causes more congestion. One may prescribe novocaine hydrochloride—a very mild anesthetic, using 5 grains of novocaine hydrochloride and 2 drams of 1 to 1,000 adrenalin to the ounce of water, to be used four or five times a day until the symptoms disappear. A certain group of cases is probably due to allergy. Dr. Ruedemann has gone into this very thoroughly. Dr. Gifford's own experience with conjunctivitis due to allergy has been very limited. One is apt to classify a condition as allergic when no other cause can be found. Before we should think seriously of allergy we should have a history of some exacerbation of symptoms on exposure to certain conditions which would give reason to think of an allergic basis. Many cases have dermatitis from cosmetics. The cases of true conjunctivitis due to allergy that he has been able to detect have been few except in cases that are really vernal conjunctivitis.

Of course we all know that the usual treatment of acute conjunctivitis is the very worst thing you can give for a patient with a vernal conjunctivitis. They do not need astringents, they need adrenalin. Sometimes it is well to use a local anesthetic for itching and to give calcium and viosterol by mouth. A few severe cases need investigation for allergy. He has not been very successful in finding the cause of vernal conjunctivitis by allergic tests. They think now at Northwestern that the routine skin tests are not of much value and they prefer tests with diluted antigen instilled in the eye.

Of course there are the cases that are due to definite chemical irritations, such as lash and hair dyes. Dyes used on the hair, get on the pillows and so into the eyes. The result is usually a dermatitis, but occasionally only conjunctivitis. Treatment of these cases after removal of the cause is with adrenalin and such a local anesthetic as novocaine. Strong astringents such as silver nitrate—which have

made it worse in his experience—should be avoided.

The most important cause of true chronic conjunctivitis is one of the pathogenic bacteria. The routine examination of secretion from all cases of chronic conjunctivitis is important. Several slides should be made—stained not only with Gram's but with Wright's or Giemsa stain. In making cultures Gifford uses epithelial scrapings, taken with a platinum spatula. Rub the surface of the conjunctiva until a layer of epithelium has been obtained, and spread like a blood film. A similar preparation is used for culture. If properly made cultures in a case are repeatedly negative, it can usually be assumed that the condition is not due to bacteria. Then other causes, such as allergy and chemical irritants must be looked for. One negative culture does not mean everything, but two negative cultures if made properly fairly exclude bacteria as the cause of conjunctivitis. One should use no anesthetic —even butyn will spoil a culture. It is important for the ophthalmologist to obtain the material himself. A laboratory technician does not know where to get the material and is afraid to touch the eye.

He does not know how common Morax-Axenfeld infection is in this region, but when in Omaha he found it very common among farmers of Nebraska and South Dakota. In Chicago this cause is infrequent. It used to be our commonest cause of chronic conjunctivitis and one must remember that it does not have to assume the typical form of angular conjunctivitis. There are three forms: (1) the typical form with redness at inner and outer angle with lid fissure and thickening of lid; (2) the more severe type that looks in some cases almost like trachoma, a velvety thickening of the upper and lower fold is present but no pannus; (3) the most common type, which is mild in objective symptoms—showing slight thickening of the lower and upper folds and a small amount of secretion present only in the morning. Perhaps a patient with such a case has been tested for glasses many times. When the diagnosis is correctly made one knows all will be cured by proper use of zinc. It is not enough to use the ordinary weak zinc at home. A 2-percent solution must be applied in the office twice a week for a month. The patient carries on with weaker solutions of zinc for several months. Something that is not very familiar to most people is chronic conjunctivitis due to pneumococci. We all know that pneumococcus is the common cause of acute conjunctivitis in America, but a certain number of chronic-conjunctivitis cases are due to it. Some of these patients have not had acute conjunctivitis—they simply, have what looks like any chronic conjunctivitis with a moderate amount of secretion. When pneumococci are found the case responds to 1-percent ethyl hydrochloride. We know that some cases we consider due to pneumococci may be due to one of the streptococci. If we investigate them carefully they can be distinguished. Gram-positive diplococci are present in smears and scrapings—and they look like pneumococci—and respond to treatment.

We have not paid enough attention to staphylococci as the cause of conjunctivitis. Thygeson has called our attention to the frequency with which staphylococci cause conjunctivitis. Allen and O'Brien have done further work on the subject. Dr. Gifford used to think that staphylococcus was pathogenic only in cases of blepharitis or styes. Thygeson also showed that the type that is pathogenic ferments mannite. It is very easy to make the cultures that ferment mannite. Such bacteria show a soluble toxin

that causes the conjunctivitis. If one does not have mannite plates it is at least suggestive to find staphylococci that are hemolytic. Even smears that show a large amount of staphylococci may suggest a staphylococcus infection especially in cases with an associated blepharitis. Unfortunately they are not so amenable to treatment as are other forms of conjunctivitis. Lederle's Toxoid No. 2 is used to immunize patients. Doses are given twice a week of .02, .04, .06, .08, .1, .2, .3, .4, .5, .6, .7, .8, .9, 1.0 c.c. A course takes about two months; some very good results are reported. Through a misunderstanding he got the idea of using Toxoid locally. It was necessary to dilute No. 2 Toxoid 1 to 100 before it would be tolerated, but it was possible to immunize the patient locally with complete relief of symptoms. He has given up the use of silver nitrate in these cases and keeps on with mild antiseptic 1 to 2,500 metaphen or any other of the mild nonirritating antiseptics

GLAUCOMA—ERRORS I HAVE SEEN AND HAVE MADE.

DR. HARRY S. GRADLE, Chicago, gave a talk on this subject.

George E. McGeary,
Secretary-Treasurer.

SAINT LOUIS OPHTHALMIC SOCIETY

January 27, 1939

DR. ROY E. MASON, *president*

INTERMITTENT OCCLUSION OF THE CENTRAL RETINAL ARTERY

DR. THEODORE E. SANDERS read a paper on this subject which was published in this Journal (August, 1939).

Discussion. Dr. Dan Myers, by invitation, said that Dr. Sanders was wise in his choice of the term "occlusion" of the retinal artery. It might be of interest to mention that this man's general vascular reactions were abnormal. After moderate exercise or subcutaneous injection of epinephrine, his blood pressure rose to 180/110, indicating a heightened vasoconstrictor response. Why this condition was more manifest in the central artery remains unanswered, but it appears logical to assume that a local lesion may have been present. It was interesting to see this man after the disappearance of these attacks. His blood pressure at rest dropped to the level of 115 systolic, and the marked lability and rise during exercise could no longer be demonstrated. Within the past three weeks he was convicted of assault and battery following a triumphant pugilistic encounter, yet this unusual exertion did not induce the symptoms of arterial occlusion. The form of straining effort which initiated spontaneous attacks was reminiscent of the Valsalva experiment in which a forced attempt at exhalation with closed glottis produces marked elevation of venous and intracranial pressures. Experimental attempts to induce retinal arterial occlusion through increasing the intracranial pressure were unsuccessful, however.

Dr. L. T. Post said he saw this patient during two of these attacks. The exercise he took to bring them on would kill an ordinary man of his age. He failed in the first three or four attempts, then finally accomplished his purpose. The pictures present the condition perfectly and leave little to add.

Dr. Carl J. Gissy said that about eight months ago he saw a man, 60 years of age, who developed an embolus that lodged in the central artery of the retina. This happened early in the morning, and he saw him about two hours later. He administered nitroglycerine, applied heavy massage to the eyeball, and at the same

time observed in the fundus blanching of the retina while making pressure on the globe. The embolus was dislodged into the inferior temporal artery with a fair return of vision and a defect in the upper part of the visual field.

Dr. William M. James said that shortly after Dr. Sanders had told him of this man, he saw a case of obstruction of the central retinal artery about an hour after the onset of the visual disturbance. The nerve head was not pale, and there was little retinal edema. The red spot had not developed in the macular area. Acetylcholine and amyl nitrite were given in the office without inducing any improvement in vision. A paracentesis was performed. When pressure was made on the globe and then sudden release there was an increase in the color of the fundus. He was under the impression that with pressure on the globe there was no obliteration of the central retinal vein and blood was forced back into the arteriolar bed of the eye. Intermittent massage was prescribed, with some improvement of the visual field.

Dr. Sanders said, in conclusion, that like Dr. Myers we are doubtful that vasodilators had much to do with bringing about the permanent disappearance of the condition and that this was probably due to a change in the reaction of the blood vessels. At no time did we note any evidence of vasodilation with the ophthalmoscope, but the reaction of the central and retinal vessels may not be identical as they are quite different anatomically. Although most of the occlusions of the central artery are obstructed by a local vascular lesion, many are due to true emboli such as cases like Dr. Gissy's in which the embolus passes into a branch while being observed. Although this patient was extremely coöperative in carrying out all our requests, the possibility of failure of the artery to reopen always was present to worry us. We were never quite sure that we would not have a permanent occlusion to treat.

Contribution to the Therapy of Some Ocular Diseases

Dr. Herman Rosenfeld, by invitation, read a paper on this subject.

Discussion. Dr. B. Y. Alvis, in opening the discussion of this paper, said he would confine his remarks to the one case, that of a young lady, 27 years of age, who had lost one eye as a result of an accident and extensive uveitis several years previously. He had seen this girl two years before the present illness, when she presented a mild uveitis and choroiditis. This attack cleared up under fever therapy, typhoid-paratyphoid administered intravenously. She went back to work and was able to earn her living and returned to him as a private patient about July 1, 1938, complaining of a blurring, although the vision was only slightly reduced. There was some increase in the vitreous opacities; no other signs of inflammation were noted with the slit-lamp. He gave her a tuberculin test at that time because he thought this was a case of tubercular uveitis. The test was negative. When the second test was negative and the vision dropped with increasing vitreous opacities and the appearance of keratitic precipitates, he gave her typhoid-paratyphoid injections, three of them, without improving the eye condition. He then gave her sulfanilamide, with no results. Her vision dropped to 3/60 when she was referred to the Jewish Hospital for study by Dr. Rosenfeld. While in the hospital she was placed on a high vitamin-B regime and the gold treatment was begun. After four treatments her vision showed definite signs of improvement. After eight weeks her vision had returned to 5/15 and the external signs of inflammation had subsided. The cells disappeared from the aqueous; some vitreous opacities remained. She re-

turned to work and he did not see her until December 30th. At that time the eye was externally quiet, the vision was almost back to its regular level, and the only signs that remained were the somewhat increased floaters in the vitreous. He would not go so far as to say that the gold therapy was the curative agent, but the fact that this violent uveitis, responding not at all to the ordinary treatments, regressed as it did, made him feel that the gold had some effect. Leprosy of the eye has also been treated with gold with good results. In the literature, while reports are not numerous, there have been other cases recorded which improved under administration of gold compounds.

Dr. W. Meinberg said his case was the second one, a girl 20 years of age. She was first seen about one year ago, when she had a chronic uveitis with vision reduced to 20/200. She was given a very thorough examination, but no focus of infection was found. He used salicylates, sulfanilamide, and typhoid-paratyphoid vaccine generally and the usual local treatment. He treated her with tuberculin although she showed no reaction to the second test dose of P.P.D. Her vision fell to counting fingers at 18 inches. He asked Dr. Rosenfeld to see her and give her gold therapy. That was two months ago and yesterday there were no cells in the aqueous. Her vision had improved to 20/200. She has received about 12 injections. He believes the gold therapy must have had something to do with this improvement.

Dr. Max Jacobs had seen two patients who came under Dr. Rosenfeld's care, one an elderly woman who had had an incipient cataract for years. Her lens opacities have remained stationary. While under Dr. Rosenfeld's care this patient's general condition was markedly improved. The other patient came under his observation while Dr. Rosenfeld was treating him for uveitis. This patient seemed to get best results from large doses of sulfanilamide.

Dr. John Green stated that he was impressed by the lack of any definite etiology in most of the cases described. Focal infection, tuberculosis, and syphilis were sought for; no thought was given to the possibility that undulant fever might be the cause. Undulant fever is very prevalent and simulates almost any disease. Laboratory confirmation is necessary before a positive diagnosis can be made. A young man with choroiditis was seen on one occasion by a very competent ophthalmologist in the South. On clinical grounds he regarded the lesion as tuberculous. Dr. Green saw the patient shortly afterward and felt that this diagnosis was correct. There was no evidence of syphilis or focal infection and all tests for tuberculosis were negative. He had a strongly positive skin test for Brucella, a Foshay index of five, and a negative agglutination test. Over a period of six months he had a course of Foshay vaccine. All signs of activity subsided, and he now presents the well-defined white scar of a healed choroidal lesion.

Aniline pencil in orbit

Dr. Leslie C. Drews gave a case report on this subject.

Discussion. Dr. J. M. Keller said that Dr. Drews's case was interesting because the fragment of indelible pencil was walled off in the orbital cavity before it caused more serious destruction, despite the fact that it had remained there for such a long time. Often these pencils contain a very soluble aniline dye which readily diffuses through the tissue, causing a protracted chronic inflammation and necrosis of the orbital tissues. If fragments of indelible pencils lie in the conjunctival sac, the whole eyeball may be stained deeply violet. The cornea shows throughout a bluish discoloration and areas of epithelial exfoliation. The iris

may show a similar staining. Cases of discoloration of the fundus to a wine-red have also been reported.

About eight years ago he saw a child two years old who had poked an indelible pencil into the right eye. His parent took him to an optometrist, who assured him that the eye would be all right. A week later when he saw the child violet-colored tears and mucus were oozing out between severely swollen lids. With a Desmarres lid retractor he was able to discover a deep blue necrotic area in the upper temporal quadrant of the retrotarsal conjunctiva between the lateral and superior rectus muscles. Several small pieces of indelible lead were removed from the wound. The whole conjunctiva and the cornea were discolored, the stain had penetrated into the anterior chamber and the whole eyeball was stained violet. The cornea was hazy and showed areas of exfoliation of epithelium; later on it was transformed into dense white scar tissue to which the temporal half of the upper lid became firmly adherent, forming an extensive symblepharon. The eyeball retained its shape.

The treatment consisted of thorough removal of the fragments of the indelible pencil and repeated irrigation with aqueous solution of tannin, 10 percent. About a year ago the upper lid was resected from the globe and skin grafting was performed. Now the young patient is wearing a well-fitted shell eye.

It has been shown that the basic aniline dye of the indelible pencil is the chemically destructive agent in these injuries. The readily soluble methyl violet is especially dangerous on account of its necrotic action. The extent of the damage depends upon the quantity, the kind of aniline dye, and the length of time it is permitted to act on the tissues. It is well to remember that tannin forms an insoluble compound, with all basic aniline dyes. Hence irrigation with a 5- to 10-percent solution of tannin has been recommended for treatment of these injuries. Hydrogen peroxide, 5- to 10-percent solution, has been advocated for removing the stain. The acid and neutral aniline dyes are practically harmless and cause little or no irritation.

Dr. B. Y. Alvis stated that he had had a patient several years ago who was standing by a desk, when the person sitting at the desk pressed a pencil on his work so strongly that the point of it broke off and flew into the patient's eye. He complained of a great deal of pain and consulted an oculist. The eye was irrigated freely and bandaged. He came to him the next morning. Upon turning the lid he found the point of the pencil in the upper cul-de-sac. The course of this case was very much like the one Dr. Keller described. There was violent inflammation of conjunctiva and cornea. The aqueous was visibly colored by the aniline. Iritis developed. The conjunctiva remained stained for days and underwent necrosis at the area of contact. Despite the fact that the point of the pencil had been removed within 24 hours the sight was much impaired, when the inflammation finally subsided, due to scarring of the cornea.

Dr. C. T. Eber said that when he saw this patient the mass under the left upper lid had surface dimensions of about one-fourth inch by one-half inch and extended backward in the orbit to an unknown depth. Before attempting any surgical measures he had an X-ray examination made which proved negative. The vision was 20/30 with each eye. The rotations of the left globe were markedly limited upward and outward; the disc margins were somewhat blurred. A blood Wassermann test was requested but the patient refused to have it made and did not return to him.

Adolph Lange,
Editor.

AMERICAN JOURNAL OF OPHTHALMOLOGY

Published Monthly by the Ophthalmic Publishing Company

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THE HERMAN KNAPP MEMORIAL HOSPITAL

With the expansion of medical knowledge and the tremendous number of instruments of precision necessary in every branch, as well as the increasingly close relationship of the specialties to the parent body, it has become more and more evident that isolated specialty hospitals no longer represent the highest type of medical service. In years past when the bulk of medical knowledge was carried in the brain of the general doctor and all the equipment in his hand bag, the specialty hospital could depend on him for the necessary general consultations. But now such consultations may lead only to dozens of special tests which cannot be carried out in the isolated hospital. Furthermore, such hospitals lack for teaching purposes the material always present on the wards of the general hospital—neurological, medical, obstetrical, and others—and if by chance patients presenting such conditions come first to the specialty hospital they cannot remain there and have these conditions followed to their ultimate conclusion.

With these things in mind it has seemed best to the directors of the world-renowned Herman Knapp Memorial Hospital to discontinue service at the present location and amalgamate with the Department of Ophthalmology of Columbia University. The funds and assets of the Knapp Hospital will be used to form the Knapp Memorial Foundation in Ophthalmology. The clinic will be taken

over by the Presbyterian Hospital and the Vanderbilt Clinic. There will be an endowment for 12 perpetual Knapp Memorial beds in this hospital, and other income from the Knapp funds will be used for graduate study, teaching, and research.

On January 1st, with the passing of the Knapp Memorial Eye Hospital at 500 West Fifty-seventh Street, a very distinguished 70-year-old institution closed. The record of this hospital, founded in 1869 by the late Dr. Herman Knapp as the New York Ophthalmic and Aural Institute, is quite remarkable in that more than 800,000 patients have been treated there—730,000 in the out-patient department without charge, and 70,000 as in-patients of whom 76 percent were treated free. Although these figures convey an idea of the quantity, they do not express the remarkable quality of the work done there. Two great teachers have headed this hospital: Dr. Herman Knapp, the founder, and Dr. Arnold Knapp, the present head. Many are the students who have worked under them and countless are those who as visitors derived invaluable information from observation of the splendid work of the staff. Most particularly the surgical clinic has been a center of learning for all America.

The hospital moved from its original site at 46 East Twelfth Street to its present location in 1913, at which time the otological department was discontinued and the name changed to the Herman Knapp Memorial Eye Hospital. Until the College of Physicians and Surgeons moved to the present Columbia-Presbyterian Medical Center in 1928, the Knapp Hospital was utilized for much of the teaching in the Department of Ophthalmology of Columbia University. The discontinuance of this connection was one of the factors that led to the present action.

Regret at the closure of the old institute would be deep indeed were it not that the continuation of Dr. Herman Knapp's plans and dreams is guaranteed by the formation of the new Knapp Foundation, to be carried out in the Ophthalmic Department of Columbia University.

Lawrence T. Post.

EDUCATING OPTOMETRISTS

Members of the Section on Ophthalmology of the American Medical Association were recently asked to declare whether they were in favor of repeal of certain resolutions adopted by the Section in the years 1935 and 1936, and dealing with the relationship between ophthalmologists and optometrists. The resolutions adopted in 1935, and subsequently approved by the House of Delegates of the American Medical Association, prohibited such professional sins as delivering addresses or courses of lectures, or giving any other form of instruction, to opticians or optometrists. The resolutions adopted in 1936 condemned alleged attempts to force ophthalmologists to instruct students of optometry.

In the past quarter of a century the movement toward legalizing and regulating the practice of optometry has spread throughout the United States and Canada. In many states the laws on the subject were adopted in spite of vigorous opposition by the medical profession.

The arguments used against such legislation have been that examination of the human eye with a view to prescribing correcting lenses is a part of the practice of medicine, and that it can only be properly performed by those whose medical training qualifies them to make accurate diagnosis, not only of ocular disease, but of its possible relations to disorders in other parts of the body.

Many unfortunate misunderstandings, especially as to the public's grasp of the distinctions involved, would have been avoided if those opticians who claimed special qualifications in refraction had promoted general and legal use of the term "refracting optician," instead of coining the name "optometrist." The former is so much more clear and straightforward than the latter that there is excellent excuse for the accusation that the title "optometrist" was created originally for the express purpose of misleading the public. The word "optometrist" has, however, by this time come into general use in spite of the fact that somewhere between 90 and 99 percent of the laity do not know the difference between an optometrist and an ophthalmologist or oculist.

If one may judge by the general average of refraction work among optometrists, the state optometric boards must be considered to have accomplished relatively little in the way of improving the standards of those upon whom they confer licenses. Thacker's excellent study of the subject (American Journal of Ophthalmology, 1939, volume 22, page 1227) indicates that, of 1,416 students of the University of Illinois, the number who had failed to be relieved of eyestrain symptoms upon wearing correcting lenses was very much greater among those who had consulted optometrists than among those who had consulted ophthalmologists.

It must be acknowledged, as is so often urged by optometrists, that the laws as to medical licensure tacitly permit general physicians, however unqualified concerning the eye and its measurements, to refract and undertake other duties which should properly come within the sphere of one who limits his practice to the eye. Nor should it be overlooked that a number of self-styled oculists are still inadequately prepared, particularly in regard to refraction work. Let us also recognize that there are a few optometrists who show praiseworthy skill in refraction, in spite of the restrictions imposed by their lack of authority to use cycloplegics.

Thacker points out that there are at present ten "Class-A" schools of optometry, as defined by the International Association of Optometry Boards at its convention in Toronto in 1934. These schools now require a four-year course. Thacker believes "that sooner or later all states will require an optometrist to be a graduate from a recognized Class-A school of optometry before he will be admitted to the state examination for license to practice."

In spite of the imposing curricula of these Class-A schools of optometry, it is still true that very few optometrists have had opportunity to develop skill in the diagnosis of ocular disease apart from refractive anomalies. To quote Thacker, "Although anatomy, pathology, and therapeutics are taught by physicians in a few of the optometry schools, by far the majority of the instruction is done by persons with a Bachelor's or Master's degree or by 'Doctors of Optometry'."

The impression among many ophthalmologists seems to be that the more an optometrist learns about ocular anatomy, function, and disease, the more dangerous he will become to the general public, "by giving it a false sense of security."

The present writer is disposed to believe that few quotations have been more greatly abused and overworked than the one about a little knowledge being a dangerous thing. If a little knowledge is a dangerous thing, does the remedy lie in attempting to refuse even a little knowledge, or, on the other hand, in helping the little to grow to greater knowledge?

Must we try to prevent optometrists

from acquiring any information at all about the human eye, or shall we encourage them to acquire more information than they now possess? Since optometric examining boards and university and other courses in optometry already exist upon a firm footing, it is obviously impractical and even ridiculous to demand that optometrists shall be deprived of the little knowledge which so many of us regard as dangerous. Quite certainly the optometrists will proceed, by one means or another, to enlarge rather than diminish the scope of their professional training.

As to the mischief arising from the public's "false sense of security," the danger was created when the optometric license was issued, and it is hardly likely to be increased by the fact that the optometrist becomes wiser instead of more ignorant. Is the danger to the public greater from an optometrist who knows how to suspect the presence of glaucoma, optic atrophy, or iritis, or greater from one who is completely ignorant of such matters? Is not the former more likely than the latter to refer his customer or patient for proper attention by an ophthalmologist?

Here and there a few ophthalmologists have accepted invitations to address meetings of optometrists, or to lecture in schools of optometry. Such engagements are not necessarily entered into for selfish advantage. Obviously, well-trained ophthalmologists are better qualified to provide instruction as to ocular disease and diagnosis than the "Doctors of Optometry" to whom Thacker attributes most of the instruction given in optometry schools.

It may reasonably be argued that by delivering educational addresses to a group of optometrists, or in giving educational lectures to students of optometry, an ophthalmologist is more likely to help than to harm the general public. Why stigmatize as unethical anyone who thus undertakes to increase knowledge among those upon whom the law has conferred a definite measure of professional status?

The problem ought to be decided purely on the ground of public interest, and not as a question of advantage or disadvantage to either optometrist or ophthalmologist.

W. H. Crisp.

GRADUATE STUDY OF OPHTHALMOLGY

The last 90 years have brought the enormous development of modern ophthalmology from a minor branch of surgery to a most important branch of modern medicine. The ophthalmoscope; general and local anesthesia; the discoveries of Pasteur, developing to a recognition of virus diseases; the localization of functions that furnish the basis of brain surgery; the forms of nerve strain called eyestrain, after Donders had pointed out their clinical importance; and a hundred other discoveries within the more limited field of ophthalmology, all have compelled graduate study. Every honest student, entering upon this branch of medical practice, has been compelled to study the new observations and suggestions that come out each year, and the similar advances in other fields of medicine and surgery press on the attention of any one who would be worthy of the title of physician or surgeon.

Graduate study has become more important and more extended than the four years of undergraduate study ever could be. But the plans and institutions for graduate study are still in a formative stage. There are no institutions devoted to it to be compared with the medical schools that stand with their prestige and facilities of hundreds of years of educa-

tional service. The methods and organizations for graduate study are just developing. And they have to recognize that the student is also a practitioner, giving most of his time to his practice. He who enters honestly upon the practice of medicine and surgery must now face a lifetime of study.

Under the stimulus of examining boards, courses of graduate study are rapidly being organized and given. Probably 20 such courses on ophthalmology are being given this year in the United States. Generally they consist of clinics and didactic lectures. These are worth seeing and hearing. It is assumed that all graduates have books that they can read, and that they also take journals. The publishers and booksellers keep up a steady supply of new books and last editions. But the real value of books and journals depends upon the way they are used. The man who reads regularly everything that interests him and connects what he reads with what he already knows, added to his daily experience, is a real graduate student. Those who catch at a new idea and do not think about it, or test it by their own experience, are merely killing time, not studying.

The man who attends a graduate course and makes no notes of what he hears, and does not read to follow up what he has been told, may be having a pleasant vacation from daily practice, but is not making himself a real student. The American Academy of Ophthalmology and Otolaryngology publishes brief condensations of the instruction courses that are given in connection with its annual meetings. These the student can use for reference to refresh his memory, or to bring up what he has heard to apply to his own cases and more fully to digest when it is most interesting to him.

The George Washington University School of Medicine, for its postgraduate course in ophthalmology, started by Dr. W. Thornwall Davis, has adopted the plan of supplying those who take the course with type-written reports of the lectures, questions, and discussions that follow. One of these, "Medical management of ophthalmic disease," by Dr. William L. Benedict of the Mayo Clinic, occupies 28 closely typewritten pages. The course on "External diseases of the eye," by Dr. John Green, occupies an equal space. The course on "Physiological optics," by Dr. W. H. Crisp, which was illustrated by lantern slides, fills 30 pages. The two volumes, 420 pages, give the current teaching of 21 of America's best known ophthalmologists. This is a new kind of teaching, and we must find by trial the best form and methods to meet the needs of lifetime students and workers. Few institutions can do what this medical school is doing. But every sincere effort in this direction will help those who engage in it. Time, winnowing the various methods tried, promises to give to our branch of specialized medicine a great and advancing development.

Edward Jackson.

BOOK NOTICES

ATLAS CLINIQUE D'OPHTALMOSCOPIE PHOTOGRAPHIQUE (Clinical atlas of fundus photographs). By H. Tille and A. Couadau. 194 pages with 202 figures. Published by Masson et Cie, Paris, France, 1939, with a preface by J. Mawas. 25 x 32 cm.

This book is designed for use by ophthalmologists in general, but particularly for the general practitioner eager for ophthalmoscopy. The atlas of black-and-white photographic reproductions of the ocular fundus fills a gap between colored drawings, as seen in the usual ophthalmoscopic atlas, and printed descriptions such as are found in the textbooks.

The plates, which vary in size—some 6½ inches, others 4½ inches, and 3 inches in diameter—are printed on smooth glossy paper that exhibits the various conditions adequately. On the pages facing the plates are to be found brief clinical histories, descriptions, and interpretations of the ophthalmoscopic findings. The legend beneath each plate is in French and English. The authors have succeeded, for the most part, in presenting fundus photographs free of annoying reflexes. An interesting and valuable method of presentation is that of piecing together photographs of various parts of the fundus so that an idea can be obtained of conditions in the periphery in relation to the rest of the fundus.

All of the usual ophthalmoscopic conditions are represented, along with a few of those more rarely seen; for example, angioid streaks, Coat's disease, and choroidal sclerosis. The pictures of angiospasm are particularly good.

Excellent as the atlas undoubtedly is, it but whets one's appetite for an atlas of colored photographs which without question someday will appear.

Derrick Vail.

GERMAN OPHTHALMOLOGICAL CONGRESS, Meeting of 1938. Paper covers, 534 pages, 207 illustrations. Munich, J. F. Bergmann, 1938.

This volume of more than usual size appears well within a year after the meeting it records. It is edited by the Secretary of the Congress, E. Engelking, of Heidelberg. In an organization that holds its fifty-second annual meeting, many new participants appear; some with familiar names now work in new fields of activity. Lindner remains in Vienna, but Lauber, who also attended the meeting, came from Warsaw. Lindner takes up some modifications of the intracapsular cataract extraction, especially describing the Verhoeff forceps for seizing the lens capsule, and the placing of stitches for the corneal incision. Lauber, who presented the first paper at the Congress, discussed the relation of the rods and cones in the retina to the isopters of the visual field. The importance of these lines of equal vision, obtained with relatively small test objects, was emphasized by Traquair at the meeting of the American Ophthalmological Society in June, 1939. But the Transactions of that meeting have not yet been published. Lauber also had a paper on the use of diathermy in the treatment of beginning choroidal growths. This paper was discussed by 12 of the members. At the Demonstration Session, 32 members of the Congress presented new apparatus, methods of examination, clinical cases, and histological or pathological specimens.

For all who read German this volume contains interesting papers and suggestions.

Edward Jackson.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP
ASSISTED BY DR. GEORGE A. FILMER

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is only mentioned in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

7
UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Cone, A. J., Moore, S., and Dean, L. W. **Relationship of paranasal sinus disease to ocular disorders.** Laryngoscope, 1939, v. 49, May, pp. 374-393. (See Section 11, Optic nerve and toxic amblyopias.)

Herman, Morris. **Argyll Robertson pupils in alcoholism.** Arch. Neurol. and Psychiatry, 1939, v. 41, April, p. 800.

Though infrequently, the Argyll Robertson pupil may occur in other conditions beside tabes or dementia paralytica. It has been observed in association with multiple sclerosis, meningitis, wood-alcohol poisoning, arteriosclerosis, encephalitis, diabetes, and tumors of the pituitary gland, pons, and colliculi.

Three cases of chronic alcoholism in which Argyll Robertson pupils were present are reported. F. M. Crage.

Jaeger, A. de. **Mobile cyst of the anterior chamber.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 116.

A case report with ten references.

McDonagh, J. E. R., and Wolff, E. **Iritis (white eye) in fowls.** Brit. Jour. Ophth., 1939, v. 23, Oct., p. 659.

Iritis ("white eye") in fowls is one manifestation of a disease complex commonly known as "fowl paralysis." It is widespread and is regarded as a subacute or chronic form of disease arising as a result of the activity of pathogenic developmental or mutation forms of bacillus coli communis. Pathologic changes in the iris, ciliary body, anterior chamber, choroid, retina, and optic nerve are discussed and illustrated by photomicrographs. The condition is most prevalent in incubated ill-fed chicks. The author seems to regard this iridocyclitis in fowls as resembling that which appears in man as a symptom of intestinal toxemia.

D. F. Harbridge.

Parry, T. G. W. **Ocular reaction to foreign protein.** Brit. Med. Jour., 1939, Aug. 19, pp. 396-397.

A case of bilateral iridocyclitis which disappeared after elimination of contact with eggs and chickens is reported. Relapse was caused both by ingestion of

and local contact with eggs. The skin test to egg white was positive. The patient was treated by desensitization, and a local flare-up followed each injection. T. E. Sanders.

Siegert, Ernst-Joachim. **Clinical and serologic diagnosis of gonorrheal iritis.** Graefe's Arch., 1939, v. 140, pt. 2, pp. 303-327.

The clinical picture of acute metastatic gonorrheal iritis is similar to rheumatic iritis, but more severe. Characteristic features are: abrupt onset with marked inflammatory symptoms, pain, severe disturbances of ocular function, formation of massive fibrinogelatinous exudate of honeycomb or flaky structure, and the strong tendency to formation of posterior synechiae which are easily torn free by mydriatics. These symptoms and the absence of nodules help to distinguish gonorrheal iritis from the syphilitic and tuberculous varieties. Most cases arise from a long-past gonorrheal infection. It is not justifiable to depend upon a history of mono-arthritis for the diagnosis of gonorrheal iritis. In the 11 cases from the Hamburg Clinic studied by the author, only three were found to have had a gonorrheal mono-arthritis. Latent urogenital disease can only occasionally be provoked into activity in these cases of iritis. One must postulate the presence of a submucous encapsulated focus from which a bacteremia arises. Serologic examination (complement fixation and precipitation reactions) is specific in a high percentage of cases. A study of the 11 examples of gonorrheal iritis from the Hamburg clinic shows the value of the "gonoreaction" in cases of iritis suspected of gonorrheal origin or in borderline undiagnosed cases. The serologic reaction proved to be a useful but not exclusive diagnostic aid. The author gives his experience with specific treatment (gonoyatren, arthigon) in patients with gonorrheal iritis, a disease which in general offers a good prognosis Charles A. Perera.

8

GLAUCOMA AND OCULAR TENSION

Biró, Imre. **Facts concerning the heredity of glaucoma.** Orvosi Hetilap, 1939, v. 83, Aug., p. 832.

Among 761 patients, glaucoma was present in more than one member of the family in 43 instances, involving 36 families. In five families only siblings were affected, all with the juvenile form. In 27 families glaucoma was present in two generations. The onset of glaucoma in the patients of the second generation occurred between the fiftieth and sixtieth years. Since in this advanced age involutionary processes might have caused the glaucoma, the hereditary factor was not taken as proved unless anticipation was present, that is, the onset occurring in the second generation at a much earlier age than in the parents. Conclusive evidence of heredity was seen in four families in which three generations were afflicted. The anticipation was very marked. Women were more often affected than men. In the same family the type of glaucoma remained always identical. In one family the onset of glaucoma simplex occurred at the age of 60 years in the grandmother, at 42 in the mother, and between the ages of 34 and 20 in six grandchildren. In a second family in which the age of the grandmother was not given, the father became affected at 58, and five daughters between the ages of 45 and 35 years. Four boys remained free of inflammatory glaucoma. In a third family both

grandparents had glaucoma, their daughters developed the disease at 38 and 35 years respectively, and a grandson at 20 years. It is of interest to note that in this family those persons who inherited glaucoma also inherited psoriasis, while those persons who remained free of glaucoma remained free of psoriasis. R. Grunfeld.

Briggs, A. H. **Familial primary glaucoma in adults.** Brit. Jour. Ophth., 1939, v. 23, Oct., pp. 649-659.

This contribution contains a survey of the scanty literature on the subject. Two glaucomatous families are discussed. All forms of glaucoma may occur in affected families but the chronic simple form seems the most frequent. The condition is transmitted by either sex, males being slightly more affected. Hereditary and nonhereditary glaucoma are likely separate conditions, but, while the exact cause is unknown, investigation seems to indicate that defective development of the angle of the anterior chamber is the probable basis. In the first family reported, of eleven individuals of the third generation three were certainly glaucomatous, two others were probably so, and two more had likely had the disease. In the second family, of nine living siblings eight were said to be glaucomatous; of these, six were examined by the author who found five glaucomatous.

D. F. Harbridge.

Fritz, A. **Hypotonic action of cold applied to the orbital region.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 127

It is stated that application of ice over the closed lids for several minutes will cause a rapid fall of ocular pressure to the extent of 5 to 10 mm. of mercury. The effect is produced not only in the normal eye, but also in the glaucomatous eye and even when it is under the influence of miotics.

Jerome B. Thomas.

Hallum, A. V. **Congenital secondary glaucoma.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1262-1266.

9

CRYSTALLINE LENS

Argañaraz, Raul. **Extraction of the opaque lens in its capsule.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Nov., p. 589.

The author discusses in detail the three principal methods of intracapsular cataract extraction. The indications and contraindications of removal by tumbling the lens, direct traction after the manner of de Grosz, and extraction by suction, together with the advantages and disadvantages of each method, are given. The difficulties and dangers of extraction "in toto" with respect to the iris, lens capsule, zonular fibers, and vitreous body are accorded full treatment. Edward P. Burch.

Argüello, D. M., and Tosi, B. **Vogt's exfoliation of the lens capsule associated with thrombosis of the temporal vein and Stähli's line.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Dec., p. 681.

In a fifty-year-old Arab biomicroscopic examination of the left eye revealed typical exfoliation of the anterior capsule of the lens, atrophy of the iris, and a horizontal pigment line with the characteristics of Stähli's line. The intraocular tension of the eye had repeatedly been found to be 17 mm. with Bailliart's instrument. The left eyeground exhibited a thrombosis of the inferior temporal vein.

Eugene M. Blake.

Berens, C., and Bogart, D. **Certain postoperative complications of cataract operations; with particular reference to a study of one thousand and four operations.** Trans. Sec. on Ophth., Amer. Med. Assoc., 1938, 89th mtg., p. 238. (See Amer. Jour. Ophth., 1939, v. 22, April, p. 463.)

Buxton, Robert. **The intracapsular extraction of cataract with forceps. Is its use justifiable?** Brit. Jour. Ophth., 1939, v. 23, Aug., pp. 505-539. (See Amer. Jour. Ophth., 1939, v. 22, July, p. 795.)

Clark, J. H. **Effect of parathyroid hormone on the permeability of the lens capsule to calcium.** Amer. Jour. Physiology, 1939, v. 126, May 1, p. 136.

Experiments were made with pig lenses immersed in solutions containing calcium. When parathyroid extract was added to the solutions in which the lenses were immersed, penetration of calcium into the lens was completely inhibited. The experiments suggest that one cause of cataract occurring after parathyroidectomy may be a greater permeability of the lens capsule to calcium in the absence of parathyroid hormone. F. M. Crage.

Cornet, Emmanuel. **Conjunctival covering with conjunctivo-episcleral and conjunctivo-corneal sutures.** Ann. d'Ocul., 1939, v. 176, July, p. 550.

The author describes briefly without illustration his method of closing cataract wounds with two conjunctivo-episcleral sutures and two conjunctivo-corneal sutures. The cornea is deliberately scarified over the area in which the conjunctivo-corneal sutures and flap are placed. John M. McLean.

Cornet, Emmanuel. **The use of my safety suture and of the demi-retrobulbar injection in lens extractions in general.** Ann. d'Ocul., 1939, v. 176, July, p. 549.

The author reviews the advantages of his "demi-retrobulbar" injection which, in addition to ordinary retrobulbar anesthesia and hypotony, also produces a paralysis of elevation without disturbing the depression. He also mentions briefly his "safety suture" which has been described previously (Amer. Jour. Ophth., 1938, v. 21, p. 1195). Peripheral iridectomy is used, but if there is the slightest suggestion of vitreous loss this is always converted into a complete iridectomy. John M. McLean.

Felfand, B. A. **Familial-hereditary ectopia lentis.** Viestnik Opht., 1939, v. 14, pt. 5, p. 72.

A review of the literature and a report of three cases (a mother and two children) of recurrent dislocation of the lens into the anterior chamber, replaceable by corneal massage. The author regards ectopia of the lens not as a local disease, but as a manifestation of a profound inherited degenerative process involving the ectodermal as well as mesodermal layers. The hereditary factor is dominant. Ray K. Daily.

Gscheidel, Ernst. **Zonular cataract and spasmophilia.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 194.

In none of seventy children who had been under clinical observation for manifest spasmophilia could zonular cataract be found. Hence spasmophilia cannot play an essential role in the etiology of zonular cataract.

C. Zimmermann.

Hamburger, Carl. **On the treatment with "glaucosan" of cases of glaucoma operated upon without success, and of complicated cataracts.** Brit. Jour.

Ophth., 1939, v. 23, Aug., pp. 557-567. (See Section 8, Glaucoma and ocular tension.)

Lindner, K. **Synopsis on cataract operations performed in 1938 with my changed technique.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 156.

After preparation of a conjunctival flap, a suture of Japanese woman's hair is placed about 0.5 mm. from the limbus in the sclera. The lens is extracted with Verhoeff's forceps through the round, maximally enlarged pupil. It has been found that with proper technique intracapsular extraction can be performed in a very high percentage of cases. The most favorable results were obtained in patients of 75 years and over. The great value of the preliminary suture is emphasized, as it prevents many severe incidents during aftertreatment. Displacement of the pupil is prevented by a double peripheral iridectomy. C. Zimmermann.

Milano, Achille. **Behavior of the aminoacid content of the crystalline lens after ultraviolet radiation.** Ann. di Ottal., 1939, v. 67, July, p. 541.

The author exposed eleven rabbits to a light source rich in ultraviolet rays and found a diminution of the azotoamines in the radiated lenses as compared to the controls. He finds an explanation of this result in photolysis of the aminoacids. (Bibliography.)

Park Lewis.

Müller, H. K. **Resorption of glucose and ascorbic acid in the lens.** Graefe's Arch., 1939, v. 140, pt. 2, pp. 258-268.

Verzàr and his pupils have demonstrated that the absorption of glucose from the intestine is inhibited by monoiodacetic acid. This led the author to test the influence of this chemical agent upon the absorption of glucose and ascorbic acid in the lenses of pigs and rabbits. He found that iodacetic acid inhibited the absorption of both glucose and vitamin C in the lens, apparently as the result of a chemical process. The permeability of the lens capsule was not altered. The senile lens contains less glucose than the lens of a young animal. The author believes this to be due to reduced glucose in the aqueous humor of an aged eye and increase in thickness of the lens capsule with reduced permeability of this structure and a reduction in the active chemical processes which influence the absorption of glucose. In conclusion, he states that the absorption of ascorbic acid as well as of glucose in the lens depends upon chemical processes which are inhibited by iodacetic acid.

Charles A. Perera.

Nicolato, A. **Etiology and pathogenesis of glaucoma following discission of aftercataract.** Arch. di Ottal., 1939, v. 46, Jan.-Feb., pp. 1-17.

Discission was performed twenty days after the primary operation, in a woman of 72 years. Acute glaucoma developed within two days, and an iridectomy was done after 15 days treatment with miotics. Twelve days later the patient died from cerebral hemorrhage. Microscopic sections of the eyeball are described and illustrated.

In a second case, described clinically, every form of antiglaucomatous treatment was unsuccessful, including iridectomy and iridencleisis. The author believes that glaucoma following discission is attributable either to mechanical factors which change the normal flow of intraocular fluids or to factors which modify the physiochemical condition of the vitreous. In

the former case, removal of the cause will restore normal tension. In the latter case, the hypertensive attack is bound up with predisposing factors whose compensation is interrupted by the discission, and in such case antiglaucomatous measures are useless.

W. H. Crisp.

Niemeyer, Waldemar. **Ectopia of the lens and arachnodactyly—syndrome of Marfan.** Trabalhos do Primeiro Cong., Brasileiro de Ophth., 1936, v. 2, pp. 533-536.

A short review of the literature and report of a case.

Noe, C. A. **Prophylactic foreign-protein therapy in cataract extraction.** Amer. Jour. Ophth., 1939, v. 22, Sept., pp. 1014-1017.

Rauh, Walter. **The lens in tetany under the influence of dihydrotachysterol.** Graefe's Arch., 1939, v. 140, pt. 2, pp. 334-353.

Holtz found a calcium-mobilizing substance in tachysterol, vitamin D_2, and toxisterol. He discovered that a stable oily solution of dihydrotachysterol had a therapeutic effect in postoperative tetany. Rauh decided to study the effects of this agent upon the development of cataract in parathyroprivic rats. He began treatment at varying intervals before and after operating, and studied the lenses with the slitlamp. His results showed the impossibility of preventing lens opacities in rats after the onset of acute tetany, but he found that the progress of these opacities could be retarded or stopped. When dihydrotachysterol was administered before operation, his studies showed that the lens was more certain to remain clear the earlier the drug was given. If treatment was started more than 48 hours in advance of operation, almost all the lenses retained their transparency. Rauh found that lens opacities seemed to clear even when these had begun before treatment. He also showed that medication had to be continued to prevent the formation or increase of lens changes. The author believes that the administration of dihydrotachysterol as a prophylactic measure offers a promising outlook in the prevention of tetany cataract in humans. He emphasizes the fact that the cataract of tetany and its treatment are quite different from senile lens opacities and their therapy.

Charles A. Perera.

Riedl, Franz. **A peculiar form of lenticular regeneration (multiple free lentoid formation) in secondary cataract in a family with hereditary perinuclear cataract.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 169.

A family was observed with apparently dominant hereditary perinuclear cataract. In five successive generations eight cases were diagnosed. The last three generations were examined and five of the seventeen living members were found to be affected. All had been operated upon previously, and peculiar formations were found on the secondary cataract, iris, cornea, and vitreous. Histologically these formations consisted of new lens fibers (so-called lentoids) originating from cells of the anterior lens capsule accumulated during operation. It is assumed that such fibers may in time become cataractous. C. Zimmermann.

Rubino, A. **Electrothermal flocculent coagulation of the aqueous extract of the crystalline lens (normal and cataractous).** Ann. di Ottal., 1939, v. 67, May, p. 391.

It is known that protein in general can be coagulated by heat. The author

endeavored to determine variations in coagulation temperature of protein soluble in normal and pathologic sera. A highly diluted protein does not exhibit visible coagulation from heat until after the addition of a few drops of acetic acid, sulphate of ammonia, or even a small amount of another colloid having an opposite electric charge. Flocculi are then formed indicating a separation of the solution into two parts.

The experiments were made with watery solutions of the crystalline lens, both normal and cataractous, and with the aqueous humor. The author propounds the theory that the electrolytic threshold of the aqueous extract of the crystalline lens is dependent on the relative proportions of proteins and lipoids. A greater amount (absolute or relative) of the lipoids together with a proportionate diminution of the soluble proteins creates and favors a notable elevation of the electrolytic threshold in cataract. Park Lewis.

Rubino, A. **Research on pressure and composition of cerebrospinal fluid of patients affected by cataract, with special regard to spinal-fluid calcium.** Boll. d'Ocul., 1938, v. 17, Dec., pp. 1004-1014.

The writer investigated the pressure and chemical composition of the spinal fluid in patients with cataract and presents the results in tabulated form. The pressure of the fluid varied within physiologic limits, but the calcium values were measurably lower in the cataract patients than in the controls. It is the opinion of the writer that changes in the calcium of the spinal fluid correspond to changes in the calcium of the aqueous. The results obtained correspond to his views of calcium metabolism in the pathogenesis of cataractous processes. (Bibliography.)

M. Lombardo.

Shevelev, M. M. **Activation of the sympathizing eye after cataract extraction on the other eye.** Viestnik Opht., 1939, v. 14, pt. 6, p. 71. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

Szinegh, Béla. **Further course of two cases of suppuration after cataract extraction under treatment with deseptyl.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 233.

Although combined treatment with heteroproteins and deseptyl had no immediate effect on vision, it promoted subsidence of the inflammatory symptoms. Later, operation for secondary cataract and other procedures could be performed with a hope of improving vision. C. Zimmermann.

Szinegh, Béla. **Two cases of bilateral ectopia of the lens in congenital syphilis.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 230.

Although ectopia of the lens is usually hereditary, these two cases show that it may be due to fetal syphilis acting through vascular damage as a developmental interference.

C. Zimmermann.

Taliercio, C. **Action of parathormone on the crystalline interchange.** Ann. di Ottal., 1939, v. 67, June, p. 451.

In parathyropriva, the accepted view of today attributes primary importance to the electrolytic equilibrium and more particularly to the chemical and physicochemical changes in the aqueous and lens, this latter being due to an increase in the pH of the blood serum with consequent diminution of the calcium content. As a result of this modification there should be an exaggerated imbibition on the part of the lens with an increase in calcium con-

tent. This would cause a precipitation of the lens protein and a diminution in glycolysis.

The present research was undertaken for the purpose of determining whether in animals (rabbits) after the extirpation of the parathyroids, the following results would ensue as other workers had asserted: an increase in the p_H of the blood, aqueous humor, and lens; an increase in the calcium content of the lens; and a lessening of the glycolysis of the cortical layers of the lens. The author had found in previous studies that the calcium content of the blood, aqueous, and lens was in normal amount. Following the method of Warburg in determining the respiratory interchange and anaërobic glycolysis of the lens cortex of the rabbit before and after administration of parathormone, variously prolonged, he found no appreciable differences in the values obtained before and after treatment.

Taking into consideration the fact that the administration of parathormone had in no way affected the calcium content of the lens, the author concluded that the lowering of the anaërobic glycolysis in the parathyroidectomized rabbits bore a direct relation to the increased calcium content of the lens itself. (Bibliography.)

Park Lewis.

Torres Estrada, A. **Cataract and acute inflammatory glaucoma.** Anales Soc. Mexicana de Oft. y Oto-Rino-Laring., 1939, v. 14, Jan.-March, pp. 25-39.

Relates three cases, in one of which the glaucoma was due to swelling of the cataractous lens, while in the other two it arose from dislocation of the lens into the anterior chamber.

W. H. Crisp.

Valle, Sergio. **Cataract operation in a case of leprosy.** Arquivos Brasileiros de Oft., 1939, v. 2, Feb., pp. 6-12.

After brief inflammatory complications, final corrected vision of 2/3. (8 references.)

Van Duyse. **Cataract and diabetes.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 157.

If sugar is found in the urine of a cataract patient the question at once arises whether the case is one of true diabetic cataract or of senile cataract occurring in a diabetic. The true diabetic cataract is subcapsular and is characterized by the following symptoms: rapid evolution, simultaneous progression in both eyes, softness, and lack of disturbance by glare. The writer discusses insulin therapy in cases of diabetic cataract destined for operation.

Jerome B. Thomas.

Van Lint and Alaerts. **Cataract following the use of a sulphamide product in a case of iridocyclitis.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 134.

Report of a case in which chronic iridocyclitis was rapidly complicated by cataract following administration of a sulphamide product. In the discussion of the report it was questioned whether the treatment had had any effect upon the evolution of the cataract. It was claimed that among the sulphanilamides, prontosil rubrum and rubiazol had caused the fewest accidents and that uliron was among the most toxic products.

Jerome B. Thomas.

Weekers, R. **Preserving the crystalline lens according to the method of De Haan-Bakker.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 109. (See Amer. Jour. Ophth., 1939, v. 22, Oct., p. 1194.)

10
RETINA AND VITREOUS

Adamantiadis, B. **Disciform degeneration of the posterior pole of the retina.** Ann. d'Ocul., 1939, v. 176, July, pp. 537-549.

This lesion is also known under the names of senile macular exudative retinitis and senile macular pseudotumor. It must not be confused with the massive exudation of Coates. Three typical cases are described, in each of which the lesion started as a hemorrhagic retinitis in the macula. The patients were not all senile, the ages being 40, 50, and 55 years respectively. All were myopic.
John M. McLean.

Basile, Giambattista. **Action of the melanophore hormone in normal subjects and in those affected by retinitis pigmentosa.** Ann. di. Ottal., 1939, v. 67, June, p. 412.

The author conducted three series of experiments to determine the effect of this hormone (obtained from the pituitary) on light perception and dark adaptation. The first group, consisting of persons with normal eyes, were exposed for ten minutes to intense sunlight, and in the meantime received instillations of the melanophore hormone in the conjunctival sac. In the second group, cases of retinitis pigmentosa, the hormone was dropped in the conjunctival sac of the right eye, the left being used as a control. In the third group, cases of advanced retinitis pigmentosa with marked hemeralopia, the hormone was injected hypodermically, and in two cases subconjunctivally. In each case improvement in the light sense and acceleration of dark adaptation were obtained. (Bibliography.) Park Lewis.

Boros, Béla. **Bilateral hole formation of the macula lutea in mongolian idiocy.** Klin. M. f. Augenh., 1939, v. 103, July, p. 91.

An infant of five months whose skin and bones were characteristic of mongolism showed incoördinate ocular movements and did not fixate. The pupils reacted sluggishly. The discs were of normal color, but both maculae showed hole formations surrounded by dark pigment. C. Zimmermann.

Burch, E. P., and Meade, J. R. **Treatment of hemorrhagic retinitis with the antihemorrhagic vitamin.** Minnesota Med., 1939, v. 22, Jan., pp. 32-33.

The authors present a case report of a 51-year-old man who had retinal hemorrhages for which they were unable to find any cause. They finally decided to use antihemorrhagic vitamin K with the result that there was considerable improvement within a few months. (2 references.)
Ralph W. Danielson.

Campos, Edilberto. **A case of retinitis punctata albescens.** Arquivos Brasileiros de Oft., 1939, v. 2, Feb., p. 5.

Description of a typical case, in a woman aged 18 years.

Charamis, J. **Cerebral and retinal circulatory disorders.** Bull. Soc. Hellénique d'Opht., 1939, v. 8, Jan.-March, p. 87.

A case report of a man who developed retinal hemorrhages a short time after a homonymous hemianopsia had cleared up. This condition was shortly followed by hemiplegia, aphasia, and death. George A. Filmer.

Chase, A. M., and Smith, E. L. **Regeneration of visual purple in solution.** Jour. Gen. Physiology, 1939, v. 23, Sept. 20, pp. 21-39.

Bleaching a visual-purple solution

with blue and violet light results in a greater subsequent regeneration than does an equivalent bleaching with light which lacks blue and violet. The regeneration is maximal when pH is 6.7. Successive regenerations in the same solution have the same velocity but form smaller amounts of regenerated substance. In vivo, the frog retina shows no additional oxygen consumption while visual purple is regenerating. T. E. Sanders.

Conti, Orville de. **Traumatic retinal angiopathy with monocular macular star.** Ophtalmos (Brazil), 1939, v. 1, no. 1, pp. 34-40.

The patient, a miner aged 43 years, had been struck in the right eye with a piece of ore. The macular star resembled that of albuminuric retinitis. The patient had an elevated blood pressure (165 mm.) and albuminuria, but the author regards traumatism as the causal agent, and the vasculorenal condition as contributing cause of the vascular changes and the macular star. W. H. Crisp.

Cordero, C. **Congenital hole of the macula and persistence of the duct of Botallo, patent ductus arteriosus.** Arch. di Ottal., 1939, v. 46, March-April, p. 85.

Beside presenting a bilateral congenital hole of the macula, the patient had a patent ductus arteriosus with variations of blood pressure between the right and left sides. There was also a variation of retinal arterial pressure which was higher on the right side. H. D. Scarney.

Euler. **A case of operative influence on angiospastic retinitis with white hypertension ("bei weissem Hochdruck").** Klin. M. f. Augenh., 1939, v. 103, July, p. 95.

A man of 58 years complaining of poor vision in the left eye showed angiospastic retinitis with choked disc. The retinitis and all subjective symptoms were relieved by subdiaphragmatic resection of the splanchnic nerve. C. Zimmermann.

Ferdinando, R. di. **Three observations of Jensen's chorioretinitis.** Arch. di Ottal., 1939, v. 46, July-Aug., p. 211.

After considering the principal symptoms of chorioretinitis of Jensen and noting the observations of other ophthalmologists, the author describes three cases of his own. On the basis of the existing bibliography and the study of his cases, the following conclusions are submitted: (1) The term "juxtapapillaris" applied by Jensen does not exclude the possibility that the focus is at some distance from the disc margin. (2) A disturbance of the blood circulation as well as a direct effect on optic-nerve sectors is present. (3) During the rise of this affection it seems likely that meteorologic variations are of some importance, and particularly during the winter. H. D. Scarney.

Filatov, V. P. **Treatment and prophylaxis of myopic chorioretinitis.** Viestnik Opht., 1939, v. 14, pt. 6, p. 18. (See Section 7, Uveal tract, sympathetic disease, and aqueous humor.)

Francois, Jules. **Angioid streaks of the retina, senile exudative macular degeneration, and elastic pseudoxanthoma of the skin** (syndrome of Groenblad and Strandberg). Bull. Soc. Belge d'Opht., 1939, no. 78, p. 99.

A case report illustrated by three fundus pictures. (20 references.)

Fritz, A. **The effects of temperature upon the retinal circulation.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 121.

Observation of the retinal vessels indicates that the circulation of the blood in that area is influenced to a certain degree by the surrounding temperature. This influence is not simply passive (linked with changes of general blood pressure), but also active, and it modifies the local arterial and precapillary tonus. Rapid changes in temperature, such as are apt to occur seasonally, tend to affect the retinal circulation; sudden hot spells causing retinal hyperemia, sudden cold spells ischemia. Considering the similarity of the retinal and cerebral circulation, it should be advantageous to foresee and avoid conditions under which sudden and severe cold might seriously affect the cerebral circulation, a highly specialized vascular mechanism, which is seriously lacking in adaptability.

Jerome B. Thomas.

Hofe, K. vom. **Ophthalmoscopic changes in diabetes.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 145.

In view of the many contradictory opinions in the literature the author examined ophthalmoscopically 322 patients in the home for diabetics at Garz. Twenty-eight had retinitis, 19 punctate hemorrhages, and 16 lipoid deposits. Most of the patients with retinitis had increased blood pressure; 32 showed vascular tortuosity, uneven caliber of the vessels, crossing phenomena, vascular new formations, or retinitis proliferans. The disc was generally not involved. C. Zimmermann.

Holt, Helen. **Retinal angiospasm.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1266-1272.

Kleefeld, G. **Retinography utilizing polarizing filters.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 112.

With this report the writer completes his researches in stereoscopic retinography by natural and by polarized light. Pictures of the fundus taken by these two methods are printed side by side for comparison. It is noted that the methods are not exclusive but complementary. Jerome B. Thomas.

Leonibus, Fernando de. **The retinal interchange in avitaminosis A.** Ann. di Ottal., 1939, v. 67, July, p. 512.

The author considers the interrelation of avitaminosis A and the development of hemeralopia as well as the effect produced on the visual purple. When exposed to light, visual purple is transformed into a yellowish substance known as visual yellow, which in turn is changed into a colorless visual white. Visual purple is regenerated from either the yellow or the white. The former change may occur in vitro, but the latter only in the presence of pigment epithelium. In the neogenesis from visual white, visual yellow is not produced as an intermediate product. Visual purple is formed directly from the visual white and its regeneration is accelerated by an increase of temperature. In solution, purple exposed to the light will lose its color afterward in the dark. The amount of visual yellow formed in the bleaching process depends on the temperature and the p_H. Visual purple is a complex protein which is acted upon by the visible rays and after its decoloration the ultraviolet rays are needed for its regeneration. (Bibliography.)

Park Lewis.

Lijo Pavia, J. **Tonoscopy and papilledema.** Rev. Oto. Neuro. Oft., 1938, v. 13, Dec., p. 281.

The first of a series of articles on this subject deals with the historical background of tonoscopy. The question of

comparative values of the diastolic and systolic pressures in the central retinal artery is considered at some length and an effort is made to explain the discrepancy in figures given by various observers. The causes of pathologic lowering of retinal arterial pressure and the absence of induced pulsation are cited. The rigidity of the central retinal artery and the relation betwen the general arterial pressure and retinal pressure in normal and pathologic instances are explained. Edward P. Burch.

Mathis, G. **Avitaminosis A and hemeralopia.** Rassegna Ital. d'Ottal., 1939, v. 8, May-June, p. 353.

Mathis employed the five-point adaptometer of Birch-Hirshfeld, with the Goldberg cone, to determine the threshold of sensation of 500 students of Torino. The subjects were submitted to an exhaustive investigation of their diet and the conditions of their home life. He found that 45 percent of the subjects were hemeralopic, with prevalence of only slight degrees of this condition, and with a constant relationship between vitamin-A deficiency and the hemeralopia. The author suggests that such mass determinations be made to determine the index of alimentary deficiencies. Eugene M. Blake.

Patek, A. J., Jr., and Haig, H. **The occurrence of abnormal dark adaptation and its relation to vitamin-A metabolism in patients with cirrhosis of the liver.** Jour. Clin. Investigation, 1939, v. 18, Sept., pp. 609-616.

Abnormal dark adaptation was observed in 19 of 24 patients with cirrhosis of the liver. In most instances only the delay of rod dark adaptation occurred, but in some cases an elevation of the cone threshold was also noted. These changes were unrelated to jaundice. They tended to persist in the presence of a diet rich in vitamin A, but administration of vitamin-A concentrates was followed by extensive improvement. The authors conclude that these findings suggest that abnormal dark adaptation in patients with cirrhosis of the liver is due chiefly to altered intermediary metabolism of vitamin A. T. E. Sanders.

Santoni, A. **Serological properties of retinal tissue.** Rassegna Ital. d'Ottal., 1939, v. 8, May-June, p. 255.

The author studied the serologic properties of the retina by the method of deviation of the complement. By studying the antigenic power of the lipoid fraction, retinal serum of the rabbit was compared with that of the ox (in toto) and with an alcoholic extract of rabbit retinal serum. Inoculation of retinal tissue in the rabbit produced some sera which reacted selectively with the homologous antigen used for immunization. The activity of these sera upon extracts of the organ prepared from the ox was slight and there was definite activity only toward watery extracts of the retina of the rabbit and the sheep. Antilipoid sera of the retina show organ specificity in that they react electively with the retinal lipoids more than with lipoids extracted from other organs. The author believes such a demonstration gives data which are valuable in comparison with other methods of extraction of lipoid.

Eugene M. Blake.

Schaffer, Károly. **The unity of the three forms of familial amaurotic idiocy on an anatomic basis.** Orvosi Hetilap, 1939, v. 83, Sept., p. 885.

On a common pathologic anatomic basis, strict selectivity of neurons binds together the infantile, juvenile, and

adult types of familial idiocy. The pathology is limited to the neuroderm, while the mesodermal elements, the vessels, and membranes are never affected.

The infantile type of familial idiocy is characterized by the ubiquity of the disease process, every ganglion cell of the central and vegetative nervous systems being affected. The ganglion cells of the retina, too, are swollen or degenerated causing thereby the cherry-red spot in the macula.

In the juvenile (Spielmeyer-Vogt) type, the ganglion cells of the cortex are affected in some areas and remain intact in others. The disease process is overwhelmingly extrapyramidal. The ganglion cells of the cerebellum, medulla, and rhombencephalon remain mostly intact. Amaurosis is generally present, but is caused by retinitis pigmentosa; the ganglion cells of the retina are never affected. In retinitis pigmentosa, too, the seat of pathology is in the neuroepithelium (rods, cones, and their cells). In this disease we have an accidental combination of two entirely different endogenous, hereditary, familial nervous disease processes; namely idiocy and retinitis pigmentosa.

In the adult (or Tufs') form we find swollen and degenerated cells in the cortex and to a lesser extent in the medulla. Near large, swollen cells we find entirely normal cells. The fundus is always normal, amaurosis is never present. R. Grunfeld.

Sorsby, A., and Oliver, J. O. **Macular coloboma: histological report.** Brit. Jour. Ophth., 1939, v. 23, Nov., pp. 724-729.

Only six histologic reports of this nature are contained in the literature. A brief review of the six cases is included. Aside from these reports on human eyes, one has been given by Hess on the right eye of a rabbit, and one by Zimmermann on the left eye of a dog. The case of a man aged 52 years is here reported with details of the histologic examination of the excised globe. Discussion as to etiology includes developmental failures, inflammation, trauma, and nevus formation; none of the theories being free from serious objection. (References, illustration.) D. F. Harbridge.

Thomson, A. M., and others. **A study of diet in relation to health. Dark adaptation as an index of adequate vitamin-A intake. 3. Relation of diet to rate and extent of dark adaptation.** Brit. Jour. Ophth., 1939, v. 23, Nov., pp. 697-723.

After a lengthy presentation of the procedures employed, it is the conclusion of the authors that neither the rate of dark adaptation nor light threshold of the fully dark-adapted eye has of necessity a close correlation with the intake of vitamin A in the diet. Considerable variation was found to occur in one individual and between individuals on the same diet. Until differences in technique and a clarification of results have been determined it is not possible to be certain how far recorded observations represent physiologic facts. (Tables, bibliography.)
D. F. Harbridge.

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Adrogué, E., and Tettamanti, J. **Papilledema.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Nov., p. 597.

This article is a review of theories on the pathogenesis of papilledema. The inflammatory and nervous-reflex theories are briefly dismissed. The

authors believe that in the state of our present knowledge the mechanical theories are in better accord with the histologic picture and clinical observations. They freely admit, however, that none of the existing theories will by itself wholly explain the pathogenesis of papilledema. Seven cases of papilledema are reported in which intracranial hypertension was present. Photomicrographs of each case are included. Eleven cases of orbital tumor, nearly all of which exhibited papilledema, are also recorded to refute Behr's theory that the zone of the papilla in which the edema occurs corresponds to the region of the optic nerve affected by the tumor.

Edward P. Burch.

Appelmans, M., and Wibo, J. **Rudimentary optic papilla and foramen.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 128.

A case report illustrated by six figures including three radiograms. (9 references.)

Balado, M., and Soriano, F. J. **Treatment of papilledema.** Arch. de. Oft. de Buenos Aires, 1938, v. 13, Dec., p. 649.

This article, which deals comprehensively with the management of papilledema, first differentiates this condition from edema of the nerve head due to causes other than increased intracranial pressure (thrombosis of retinal veins, neuroretinitis, optic neuritis). The authors fully discuss choked disc due to brain tumor, pseudotumor, arterial hypertension, and syphilis. The value of Balado's procedure of iodopneumoventriculography in the localization of intracranial lesions is stressed. The conclusion is reached that if the tumor is localized and amenable to surgical extirpation it should be removed. If removal is not possible a temporal decompression is advisable. Indiscriminate lumbar puncture in brain tumor or suspected tumor is to be avoided.

In arterial hypertension, lumbar puncture may be used in conjunction with the usual medical treatment. In syphilis, lumbar puncture along with antisyphilitic measures is often desirable and if necessary trepanation may be carried out. The various conditions are illustrated by appropriate case reports. Edward P. Burch.

Biffis, Andrea. **Coloboma of the optic papilla.** Ann. di Ottal., 1939, v. 67, July, p. 522.

Congenital optic-nerve excavations (psuedoglaucoma) have been recorded in numerous instances. In the case described, the physiologic cupping occupied almost the entire disc with clearly defined margins and a small rim of normal nerve tissue around the entire circumference of the papilla. The depression had a depth of about 2 mm. as determined by skiascopy. The affected eye exhibited divergent concomitant strabismus to the extent of 30 degrees. The pupil was of normal size and reaction. Central vision was absent; peripheral temporal vision was 1/50 with a myopia of 7 diopters and astigmatism of 1 diopter with the rule as determined by skiascopy. Vision in the right eye was 10/10, and the visual fields were unaffected. When monocular, the predilection shown by these malformations for the left eye is so frequent as to seem hardly fortuitous. (Plate, bibliography.) Park Lewis.

Bonhomme, F. and Barac, G. **Alteration of the optic nerve and pernicious anemia.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 143.

The writers report a case of pernicious anemia with central optic neuritis of both eyes, in which there developed a simple optic atrophy with central scotoma. They express the conviction that one should conduct a careful study of the blood in cases of optic-nerve trouble of indeterminate origin. One should not fail to recognize pernicious anemia and, thanks to the method of Whipple, it should be possible to improve the patient's condition considerably. (12 references.)

Jerome B. Thomas.

Cone, A. J., Moore, S., and Dean, L. W. **Relationship of paranasal sinus disease to ocular disorders.** Laryngoscope, 1939, v. 49, May, pp. 374-393.

The authors saw four cases of retrobulbar neuritis and one case of iritis in which sinus disease was thought to be the causative factor. The value of laminographs or body-section radiographs in establishing the relationship of posterior sinus disease to retrobulbar neuritis is emphasized.

T. E. Sanders.

Fontana, Giuseppe. **A probable case of opticochiasmatic arachnoiditis.** Arch. di Ottal., 1939, v. 46, July-Aug., p. 251.

The ocular disease began twenty days before the patient came under observation. Headache and diminution of vision were the chief complaints. Objectively there was seen a marked pallor of both optic-nerve heads. Field examination showed a narrowing of both fields with irregular limitation for white and colors. Nothing was revealed by radiologic, otologic, or neurologic examination. Spinal-fluid tests indicated marked increase in the pressure and protein. Because of the above findings the author considered an intracranial lesion, particularly at the chiasm. He believes that in a differential diagnosis one must consider lesions occurring in the diencephalic-hypophyseal area, the retrobulbar neuritides, and serous meningitis. The diagnosis was substantiated by the improvement of the patient following three months of iodine therapy. Surgical intervention was considered but the author felt that medical treatment should first be instituted.

H. D. Scarney.

Holloway, L. W. **Quinine amblyopia in children.** Jour. Florida Med. Assoc., 1938, v. 25, Oct., p. 167.

The literature is reviewed briefly and four cases reported. The author believes that children are more susceptible than older persons, and that their degree of recovery is markedly less.

George A. Filmer.

Larsen, Victor. **Parenchymatous syphilitic keratitis and syphilitic atrophy of the optic nerve treated with sulfosin.** Brit. Jour. Ophth., 1939, v. 23, Sept., pp. 585-622. (See Section 6, Cornea and sclera.)

Lijo Pavia, J. **Tonoscopy and papilledema.** Rev. Oto.-Neuro.-Oft., 1938, Dec., p. 281. (See Section 10, Retina and vitreous.)

Philps, Seymour. **The ophthalmological complications of leontiasis ossea.** Brit. Jour. Ophth., 1939, v. 23, Nov., pp. 729-738.

Creeping periostitis of the bones of the face and skull, and a diffuse osteitis of the bones of the face and skull are illustrated by case presentations as being the two types of hyperostosis. Historical cases are first reviewed, the author then recording the one to come under his own observation, a male aged 33 years, seen for the first time in July, 1938. The patient attributed his ail-

ment to the results of a motor accident fifteen years previously at which time he had suffered a heavy pressure on his head. However, photographs taken eighteen years previously indicated that a marked bony deformity was present at that time. The patient submitted to operation for the removal of a strip of bone, and this procedure was followed by marked general improvement and freedom from headache. However, the headaches recurred after a blow on the head, and the swelling on the head had increased when the patient was last seen. Atrophy of the left optic nerve was feared. (Illustrations, bibliography.) D. F. Harbridge.

Vidal, F., and Courtis, B. **Microscopic anatomy of the optic nerve.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Dec., p. 668.

A detailed discussion of the histologic structure of the optic nerve with particular emphasis on the neuroglial elements. (Photomicrographs.)
Edward P. Burch.

12

VISUAL TRACTS AND CENTERS

Leary, J. L., and Bishop, G. H. **Margins of the optically excitable cortex in the rabbit.** Arch. of Neurology and Psychiatry, 1938, v. 40, Sept., p. 482.

Using rabbits, the extent of the optically excitable cortex was measured by applying single maximal electric shocks to the cut end of the optic nerve and recording the location of the resulting stimuli in the contralateral cortex. Histologic studies of the cortex were made as controls for the physiologic records. (Illustrations.)
George A. Filmer.

Leonibus, F. de. **Visual function of the cortical visual sphere in rabbits.** Rassegna Ital. d'Ottal., 1939, v. 8, May-June, p. 274.

The author studied the respiratory exchange and the anaërobic glycolysis of the cortical visual area by Warburg's technique (direct method), comparing the results in a series of rabbits kept in the dark for five days with those in a series which was adapted to diffuse daylight. No appreciable difference was found in respiratory activity but a measurable increase of anaërobic glycolysis was noted in the dark-adapted animals. Eugene M. Blake.

Sanford, H. S., and Bair, H. L. **Visual disturbances associated with tumors of the temporal lobe.** Arch. Neurol. and Psychiatry, 1939, v. 42, July, p. 21.

The experience of the authors with many cases of this type leads them to several conclusions, the most important of which are as follows: (1) Homonymous hemianopsia is the most common visual-field defect produced by tumors of the temporal and occipital lobes. (2) Quadrantic anopsias are produced much more frequently by tumors of the temporal lobe than by tumors of the occipital lobe. (3) Normal fields occur with approximately the same frequency in cases of tumor of the temporal lobe and in cases of tumor of the occipital lobe. (4) Neither the presence or absence of choked discs nor the equality or inequality of the degree of choking in the two eyes are of any clinical value in deciding on which side of the brain a tumor of the temporal lobe is located. (5) Visual disturbances other than field defects are of no value in helping to diagnose or lateralize tumors of the temporal lobe.
Ralph W. Danielson.

Vogt, Alfred. **The riddle of amblyopia ex anopsia.** Klin. M. f. Augenh., 1939, v. 103, Sept., p. 291.

Vogt's reasons for placing the seat of amblyopia ex anopsia in the brain, and not in the eye, are that the color and light sense of the whole retina of the amblyopic eye are preserved, and the retina shows no ophthalmoscopic or anatomic imperfections. The possibility of improving the vision by early visual exercises proves the existence of dormant function in the macula and in the intact retina as a percipient organ. The difference in behavior between the periphery, the vision of which is not affected, and the center suggests that the seat of the condition lies in the brain, where periphery and center are also represented independently. The cerebral seat of the amblyopia existing after operation in individuals who had been blind from congenital cataract is discussed. Comparable conditions of dormant function as to important cerebral activities are found in the cerebral speech centers, in right-handed and left-handed persons. After destruction of the functioning side of the brain, the symmetrically opposite center is awakened, and is stimulated to activity by use. The author cites a case in which occlusion of the central retinal vein of the visually active left eye forced into use the converging and extremely amblyopic right eye, useful macular function being obtained in spite of the mature age (44 years) of the patient.

C. Zimmermann.

13

EYEBALL AND ORBIT

Alagna, G. **Case of Hand-Schüller-Christian syndrome.** Rassegna Ital. d'Ottal., 1939, v. 8, May-June, p. 287.

Alagna reviews thoroughly the three types of generalized primary lipoidosis, as classified by Epstein. He then stresses the ocular symptoms, pointing out the diagnostic difficulties. He gives a review of the known etiologic factors, symptomatology, and pathologic anatomy of the Hand-Schüller-Christian syndrome, and describes a case in a woman of 43 years, who presented the classical triad of symptoms (skeletal alterations, diabetes insipidus, and exophthalmos). The author points out in particular that ophthalmologists should be well informed on this condition, since they are frequently the first to be consulted. The article gives an excellent review of this rare disease. (5 figures.) Eugene M. Blake.

Appelmans, M., and Puffet, E. **Meningocele penetrating the optic foramen.** Bull. Soc. Belge d'Opht., 1939, no. 78, p. 138.

A case report with two radiograms and eight references.

Castro Lima, O. de. **Syndrome of the orbital apex and paratrigeminal syndrome of the ocular sympathetic.** Arquivos Brasileiros de Oft., 1938, v. 1, Dec., pp. 129-134.

A woman who some years earlier had undergone treatment for syphilis complained of frontal pain of gradually increasing severity. After a week or so the pain was accompanied by attacks of vomiting, sensations of heat and cold, and other sensory disturbances. Examination showed paralysis of the left upper eyelid and of all ocular movements. The vision of the left eye was reduced to 1/6. There were signs of disturbance of the ocular sympathetic, including enophthalmos, hypotony, and the neuralgic disturbance already mentioned. The pupil was contracted and did not react to light. Treatment with

neosalvarsan and bismuth caused steady improvement. The last symptoms to disappear were the enophthalmos, the miosis, and the hypotony. The vision returned to normal. The case is interpreted as representing the syndrome described in the title.

W. H. Crisp.

Cavallacci, G. **Syndrome of Laurence-Biedl with bilateral congenital microphthalmos of moderate degree. Pathogenic considerations.** Arch. di Ottal., 1939, March-April, v. 46, p. 111.

A typical case of Laurence-Biedl syndrome, characterized by dystrophia adiposogenitalis, polydactylism, and retinitis pigmentosa, and with an associated bilateral congenital microphthalmos of moderate degree, is described. The author is of the opinion that a definite pathologic analogy exists between the typical Laurence-Biedl syndrome and the hypophyseo-diencephalic syndrome with congenital microphthalmos, as described in a previous paper (Amer. Jour. Ophth., 1939, v. 20, p. 968).

H. D. Scarney.

Cohen, J. T., and Fisk, C. **Chronic idiopathic xanthomatosis of the Hand-Schüller-Christian type, with special reference to the oral manifestations.** Jour.-Lancet, 1939, v. 59, May, pp. 192-197.

Three cases of this condition are reported, all of which had marked exophthalmos. One case had a typical buphthalmos which required operation.

T. E. Sanders.

Kassay, Dezsö. **An instructive case of orbital cyst.** Orvosi Hetilap, 1939, v. 83, Aug., p. 838.

A small growth upon the floor of the left orbit of a 32-year-old farmer caused protrusion with upward and inward displacement of the eye. X-ray pictures, taken following the injection of a 40-percent lipoid solution into the Highmore antrum, suggested that a tumor was growing into the orbit from the antrum. At operation, however, there was removed a thick, stiff-walled cyst whose concentrically lamellated structure closely resembled that of an echinococcus cyst. R. Grunfeld.

Licheri, Giovanni. **A rare case of exophthalmos.** Arch. di Ottal., 1939, v. 46, Jan.-Feb., pp. 71-84.

The patient, a man of 30 years, was a chronic sufferer from migraine. In one of his attacks, he experienced a sudden intense pain in the left orbit, and within two hours the eyeball protruded decidedly. There was also double vision. Exploratory retrobulbar puncture showed the exophthalmos to be due to hemorrhage, which the author interprets as definitely connected with the migraine. (References.)

W. H. Crisp.

Mutch, J. R. **Description of a new proptometer.** Brit. Jour. Ophth., 1939, v. 23, Oct., p. 677.

This instrument has a central plunger which is attached to a sliding millimeter scale. Two fixed points, at the center of the upper and lower orbital margins, are selected and bridged by a curved metal band. The plunger passes through this band. In order to measure the prominence of an eye the plunger is allowed to rest upon the closed lids, the millimeter scale indicating the amount of enophthalmos or exophthalmos present. The author found upon testing many healthy adults that in 90 percent the reading was zero. (Illustrations.)

D. F. Harbridge.

Radnot, Magda. **Pathology of lymphangioma of the orbit.** Graefe's Arch., 1939, v. 140, pt. 2, pp. 328-333.

Since Förster's description of the first case of lymphangioma of the orbit in 1886, 26 examples have been reported. The author now describes two cases, one in a three-year-old boy, the other in a 34-year-old woman. In the latter, unilateral exophthalmos had been noted since the age of six years. A study of the microscopic preparations led to the conclusion that the lymphatic tissue in these tumors is not of congenital origin, but is secondary to the stimulus of hemorrhages and their absorption. Charles A. Perera.

Russo, M. **A contribution to the histogenic interpretation of orbital angiocavernoma.** Ann. di Ottal., 1939, v. 67, June, p. 463.

A relatively small number of cases of angiocavernoma of the orbit have been reported. A growth which was accompanied by marked monocular proptosis was removed with conservation of the globe. The author concludes from his histologic examination that the growth was a hyperplasia rather than a tumor formation. (Bibliography, 1 plate, 3 figures.) Park Lewis.

Schwartzman, J., and Maffia, A. **Congenital bilateral anophthalmos.** Arch. of Pediatrics, 1939, v. 56, April, p. 240.

The authors review the literature regarding the formation and etiology of this condition. A case report is given in which the anophthalmos was associated with cleft palate and polydactylism. Treatment, of course, is only cosmetic. (19 references.)

Ralph W. Danielson.

Smelser, G. K. **The histology of orbital and other fat tissue deposits in animals with experimentally produced exophthalmos.** Amer. Jour. Path., 1939, v. 15, May, pp. 341-352.

The work reported in this article is a continuation of that which Smelser has been doing for several years in an attempt to determine the cause of exophthalmos. In this experiment he examined the fat, not only from the orbit but also from the axilla and neck, and about the kidney, uterus, and ovaries. By injecting extract of the anterior lobe of the pituitary gland, he was able to produce exophthalmos and marked edema of orbital fat and connective tissue. All of the fat showed some edema, but of a lower degree than in the orbit. The difference in degree of edema produced may be due to difference in structure of the orbital fat. (18 references, 8 photomicrographs.)

Ralph W. Danielson.

Smelser, G. K. **The role of the cervical sympathetic ganglia and Müller's orbital muscle in experimental exophthalmos.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1201-1209.

Wilber, I. E. **Human cyclopia with associated ocular anomalies.** Amer. Jour. Ophth., 1939, v. 22, Oct., pp. 1120-1126.

14

EYELIDS AND LACRIMAL APPARATUS

Bardanzellu, Tomaso. **Correction of ptosis by Nida's method.** Rassegna Ital. d'Ottal., 1939, v. 8, July-Aug., p. 449.

The article contains nothing new, merely citing one case and pointing out the advantages of Nida's method, namely attachment of the superior rectus to an opening in the tarsus. (2 figures.) Eugene M. Blake.

Hirose, K. **Congenital coloboma and incomplete development of the meibomian glands.** Arch. d'Opht., etc., 1939, v. 3, Aug., p. 673.

Except for a single Japanese case report, there is nothing in the literature regarding anomalies or absence of the human meibomian glands. By means of transparent preparations (glycerin and caustic potash) of the upper and lower lids taken from cadavers, the author has been able to study the number, size, and interspacing of the normal glands. For example, the average number of glands in the normal upper lid is 37.4, in the lower lid 27.8. Absence or incomplete development of the meibomian glands was found in 16 cases. The question of atavism is posed to explain these findings. (Photographs, tables.)

Derrick Vail.

MacGillivray, A. M. **Infantile dacryocystitis treated by surgical diathermy.** Brit. Jour. Ophth., 1939, v. 23, Sept., pp. 630-631.

This observer has satisfactorily treated such cases by opening the sac to its full length, retracting the edges of the wound, and, with a thin blunt-pointed probe attached to a diathermy apparatus, cauterizing the sac together with the entrance of the duct into the nose. No sutures are used, the wound being allowed to granulate, and a neat scar usually forms within a week.

D. F. Harbridge.

Mancilla Herculano, Cueto. **Extirpation of the lacrimal sac.** Anales Soc. Mexicana de Oft. y Oto-Rino-Laring., 1938, v. 13, July-Dec., pp. 42-48.

Detailed description of the classical procedure.

Meyer, F. W. **Bilateral gummatous syphilis of the lacrimal glands.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 200.

A case of very rare bilateral gummatous syphilis of the lacrimal glands is demonstrated with histologic findings. Attention is called to the uncertainty of the Wassermann reaction in such cases, and to the significance of defect of the lacrimal glands in the etiology of filamentous keratitis.

C. Zimmermann.

Miller, Hugh. **A new surgical technique for dacryocystitis.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1278-1280.

Wood, M. A. **A study of methemoglobin-producing organisms in ocular inflammations.** Amer. Jour. Ophth., 1939, v. 22, Oct., pp. 1111-1119.

Yrys, J. M. **A case of chronic ulcerative blepharoconjunctivitis.** Anales Soc. Mexicana de Oft. y Oto-Rino-Laring., 1938, v. 13, July-Dec., pp. 40-41.

In a girl of three years, the severely suppurative and ulcerative condition had existed since the age of eight days. Culture pointed to staphylococcus albus. A cure was effected in one month by instillation of a 3-percent solution of sulphate of magnesium every three hours, followed each time by a 5-percent solution of mercurochrome.

W. H. Crisp.

15

TUMORS

Agatston, S. A., and Gartner, S. **Melanosarcoma of the ciliary body and iris.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1273-1278.

Argaud, R., and Calmettes, L. **Schwannoma of the lower lid.** Arch. d'Opht., etc., 1939, v. 3, Aug., p. 690.

A tumor the size of a hazelnut was found in the lower lid of a woman of 77 years. It was not inflamed or tender. No regional adenopathy was found. Following complete removal, the mass was fixed and stained by different methods. Microscopic study showed it to be a plasmodial hyperplasia with secondary fibrillation of the sheath of Schwann, separating palisaded nuclei. (Illustration, bibliography.)

Derrick Vail.

Benedict, W. L., and Love, J. G. **Cavernous hemangioma of the orbit with hyperostosis.** Amer. Jour. Ophth., 1939, v. 22, Oct., pp. 1149-1151.

Borsello, Giuseppe. **Bilateral retinal glioma with atrophy of one eye and prolapse of the anterior segment of the other.** Rassegna Ital. d'Ottal., 1939, v. 8, July-Aug., p. 467.

It is rarely possible to study histologically an eye which has had a retinoblastoma for a long time because in most such cases the eye is removed early. Borsello first diagnosed a bilateral retinoblastoma in a two-year-old child in July, 1937. The patient did not return until December, 1938. At this visit the right eye was enlarged and markedly exophthalmic, the cornea infiltrated and partially destroyed, the sclera thinned, and the conjunctiva injected. The left eye was atrophic and free from inflammatory reaction. The cornea was opaque in an annular zone contiguous to the limbus, the pupil dilated, and the lens opaque. The right eye was removed. The fact that there was no evidence of invasion of the orbit or of metastasis, is explained by the author on the basis of inflammatory reaction in the cornea, perforation, and therefore progression of the growth forward. The histologic changes are reported in detail and the literature is reviewed. (2 figures.)

Eugene M. Blake.

Colombo, Giuseppe. **Primary angioma of the plica semilunaris.** Ann. di Ottal., 1939, v. 67, July, p. 550.

The author describes a case of primary angioma of the plica semilunaris. He found only six cases reported in ophthalmic literature. The histology involved and the genesis of the neoplasm are discussed. (Bibliography, 1 plate, 4 figures.)

Park Lewis.

Cordero, Celso. **A case of primary medullary epithelioma of the caruncle.** Arch. di Ottal., 1939, v. 46, Jan.-Feb., pp. 18-39.

The patient, a man of 73 years, belonged to a family in which it was possible to place on record eight cases of death from carcinoma out of 32 members. A genealogical table is given. In the case now reported in detail the tumor recurred after excision. Microscopic examination showed greatly malignant characteristics, with abundance of karyokinesis, intensity of degenerative metamorphosis, scarcity of stroma and of blood vessels, and a tendency to infiltrate normal surrounding tissues. (5 figures, 6 photomicrographs.)

W. H. Crisp.

Corrado, Antonio. **Lymphangioma of the caruncle.** Rassegna d'Ottal., 1939, v. 8, July-Aug., p. 454.

Corrado describes a rare example of lymphangioma of the caruncle, circumscribed in form. This is the second case to be described in the literature. In the author's case the lymphangioma occurred in the right eye of a nine-year-old boy, and was considered congenital, there being no history of preceding injury or irritation. It was apparently

benign as there was no sign of recurrence at the end of a year and a half. (3 figures.) Eugene M. Blake.

Fontana, G., and Remaggi, P. L. **A rare case of lipoma in the corresponding region of the lacrimal sac and of the internal commissure of the eye.** Arch. di Ottal., 1939, March-April, v. 46, p. 127.

A rare case of lipomas in the region of the lacrimal sac and the internal commissure of the eye occurring in a baby of nine months is described. The lipomas were removed and histologic examination confirmed the clinical diagnosis. H. D. Scarney.

Laborne Tavares, C., and Elejalde, P. **A case of dermoid cyst of the orbit.** Ophtalmos (Brazil), 1939, v. 1, no. 1. pp. 24-33.

In a negro aged 27 years, there had for three years been gradually increasing protrusion of the right eye, with diminution of vision. The tumor, of the size of a walnut, had pushed the globe downward 4 mm. and forward 3 mm. Deformity of the cornea had caused an astigmatism of 12 diopters. (6 illustrations.) W. H. Crisp.

Meirelles, Guilherme. **Malignant lymphocytoma of the orbit.** Ophtalmos (Brazil), 1939, v. 1, no. 1, pp. 57-61.

A small tumor was removed from the upper outer border of the left orbit, in a woman of 54 years. A recurrence was removed a year later and the patient was subjected to brief radiotherapy. Three months later there had apparently been no further recurrence. (3 illustrations.) W. H. Crisp.

Roberts, A. W. **Intraocular tumors.** Jour. Arkansas Med. Soc., 1939, v. 35, Jan., p. 145.

This article is limited to discussion of two of the more common intraocular tumors, melanosarcoma of the choroid and retinoblastoma. Two cases of each of these types are recorded and the diagnosis, progressive stages, and pathology are discussed. The diagnosis of melanosarcoma may become very difficult when the mass is covered by a serous detachment. Not too much value can be placed on elevation of tension for it may remain normal. Early enucleation is strongly advised in the case of either type. F. M. Crage.

Santa Cecilia, J., and Elejalde, P. **Meningioma of the fronto-orbital region.** Ophtalmos (Brazil), 1939, v. 1, no. 1, pp. 9-23.

The tumor, in a woman aged 18 years, had seemingly developed in the course of a few months. There was protrusion of both eyeballs with destruction of the frontal bone. The patient died of suppurative meningitis, pneumococcic in character. Necropsy showed extensive destruction of the frontal bone, of other bones of the upper part of the face, and of the base of the skull, and there had been some invasion of the anterior pole of each cerebral hemisphere. Histologically the tumor showed a number of cell types, with some formation of bone. (13 illustrations.) W. H. Crisp.

16

INJURIES

Castelli, Adolfo. **A rare traumatic lesion of the optic nerve.** Rassegna Ital. d'Ottal., 1939, v. 8, July-Aug., p. 426.

Various types of injury to the optic nerve from head traumatisms are described. The author then relates the case of a nine-year-old boy who fell from a bicycle, striking the right eye against the handle bar with sufficient force to render him partially uncon-

scious. Fundus examination showed intense pallor of the retina with marked edema, except for a strip of normal retina running from the disc to the macula (the region supplied by a cilioretinal artery). The disc was pale and the borders blurred. The inferior temporal artery showed segmentation of blood and a few hemorrhages were scattered about. The retinal changes gradually cleared, leaving an atrophic nerve. The changes are ascribed to indirect trauma of the optic nerve through forced rotation of the globe, the lesion being vascular and nervous and situated behind the globe. (5 figures.) Eugene M. Blake.

Gandara Yañez, Enrique. **Action of light on the eyes and occupational risks.** Anales Soc. Mexicana de Oft. y Oto-Rino-Laring., 1939, v. 14, Jan.-March, pp. 7-15.

The author reviews the subject, presenting a series of rather familiar conclusions regarding the effect of infrared rays upon conjunctiva and lens, the effect of ultraviolet light in general and especially upon welders, and the results of electric short circuits.

W. H. Crisp.

McClelland, C. C. **Removal of BB shot with giant magnet.** Jour. Michigan State Med. Soc., 1939, v. 38, March, p. 216.

BB shot were formerly made of lead; today they are made of steel and some are copper-coated. A description is given of the removal by giant magnet through the wound of entry of a BB shot which had lodged in the vitreous chamber. Vision returned to normal, the lens remained undamaged, and there had been no retinal detachment nine months after removal of the foreign body. F. M. Crage.

Passos, T. M. **Ocular traumatisms in labor accidents.** Trabalhos do Cong. Brasilerio de Ophth., 1936, v. 2, pp. 537-540.

The author reports that in 10,577 cases of accident observed in one of the large insurance companies in São Paulo, 14.2 percent were ocular injuries, most of them from corneal foreign bodies. The author discusses the treatment in detail. Allusions are made to the Brazilian legislation as to compensation cases. Ramon Castroviejo.

Rizzi, C. **Free motile cyst in the anterior chamber of traumatic origin.** Rassegna Ital. d'Ottal., 1939, v. 8, May-June, p. 343.

A 15-year-old boy was struck in the left eye by a chip of glass, which caused a small penetrating wound of the cornea. Examination showed a practically full anterior chamber in which there was a yellowish, freely movable, spherical body about 1 mm. in diameter, which tended to remain in the upper part of the chamber. A few weeks later symptoms of irritation appeared and the cyst was removed by an incision in the cornea, but was unfortunately lost during the operation. In the absence of any injury to the iris, the author concludes that the body was a cyst of traumatic origin arising from the corneal epithelium, but he does not explain how it could develop immediately after the injury. (One colored figure.)

Eugene M. Blake.

Rodriguez-Roda, J. R. **Chemical conjunctivitis.** Rassegna Ital. d'Ottal., 1939, v. 8, July-Aug., p. 417.

The author reports 65 cases of conjunctivitis occurring among sailors who were thrown from a boat into a sea upon which a heavy coating of petroleum was present. Considerable varia-

tion in severity of symptoms was observed. Palpebral and subconjunctival hemorrhages were so frequent that he suggests the term "ecchymotic chemical conjunctivitis." Warning is given against the use of strong antiseptic solutions as they delay healing, while methylene blue was found to be bland and to possess an ischemic action. Two-thirds of the cases presented a slight hypotension which persisted for four or five days.

Eugene M. Blake.

Shuman, G. H. **The paraffin-film treatment of burns of the eyelids.** Pennsylvania Med. Jour., 1939, v. 42, May, p. 907.

In this article the author limits his work to a consideration of the immediate local treatment of burns more severe than first-degree erythemas. After cleansing the surrounding skin and carefully removing the skin tags, 2-percent aqueous solution of mercurochrome is applied to the burned area. To prevent contraction of scars it is considered very important to put the burned area on the stretch before applying the mercurochrome. When dry, alternate layers of melted paraffin and gauze are applied over the closed lids. Cotton, patch, and adhesive for fixation are next applied. The author concludes that the mercurochrome helps in desiccation of the wound, is not irritating, and, unlike tannic acid, does not kill viable tissue. The wax dressing itself affords freedom from pain.

F. M. Crage.

Terry, T. L., Chisholm, J. F., Jr., and Schonberg, A. L. **Studies on surface-epithelium invasion of the anterior segment of the eye.** Amer. Jour. Ophth., 1939, v. 22, Oct., pp. 1083-1108.

17

SYSTEMIC DISEASES AND PARASITES

Amendola, F. **General considerations of ocular complications in Hansen's disease: neuroparalytic manifestations.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 527-532.

The author enucleated a completely blind and painful eye of a patient affected with Hansen's disease. Corneal staphyloma, iridocyclitis, and scleral involvement were present. Microscopic examination revealed the posterior segment of the eye to be unaffected. The cornea was found to have interstitial keratitis with marked lymphocytic and plasmocytic infiltration. The ciliary body and the iris also presented considerable lymphocytic and plasmocytic infiltration, as well as Virchow's leprous cells. The sclera revealed foci of infiltration and suppuration. Bacilli of Hansen were found in the cornea, iris, and ciliary body. Another case of leprosy in which the pupils showed a difference in size is reported to prove that difference in size of the pupils is not pathognomonic of syphilitic conditions. Three more cases are reported of keratitis secondary to lagophthalmos due to leprous paralysis of the orbicularis. The symptoms due to lagophthalmos disappeared in all the patients after tarsorrhaphies. The following conclusions are drawn by the author after study of these cases: (1) The anterior segment of the eye is most often the site of pathologic manifestations of Hansen's disease. (2) In all patients affected with lagophthalmos tarsorrhaphy operations should be performed as soon as possible to avoid serious complications due to exposure. (3) Hansen's disease may be a cause of difference in size of the pupils. (4) Periph-

eral nervous lesions are the ones most frequently found in Hansen's disease.
Ramon Castroviejo.

Burky, E. L., Thompson, R. R., and Zepp, H. M. **The role of brucella in human and animal ocular disease with special reference to periodic ophthalmia in horses.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1210-1217.

Hansel, F. K. **Allergy in otorhinolaryngology and ophthalmology.** Laryngoscope, 1939, v. 49, May, pp. 323-373.

The author reviews the recent literature on allergy as related to otorhinolaryngology and ophthalmology. The relative interest in allergy of the two specialties is demonstrated, as 93 articles on otorhinolaryngology, but only 14 on ophthalmology are discussed.
T. E. Sanders.

Klauder, J. V., and Cowan, A. **Corneal examination and slitlamp microscopy in diagnosis of late congenital syphilis, especially in adults.** Jour. Amer. Med. Assoc., 1939, v. 113, Oct. 28, p. 1624.

Six cases are reported to illustrate the importance, in all patients regardless of age, of inspection of the cornea by slitlamp microscopy in the exclusion of congenital syphilis. (2 illustrations, discussion.) George H. Stine.

Paula Santos, B. **Photographic documentation of a cysticercus in the vitreous.** Arquivos Brasileiros de Oft., 1938, v. 1, Dec., p. 124.

A plate containing 12 fundus photographs (made by Belfort Mattos) shows in somewhat hazy outline a series of changes in the position of the scolex. The details which may be studied deliberately in such photographs might easily be overlooked during ophthalmoscopy. W. H. Crisp.

Sanders, T. E. **The ocular Shwartzman phenomenon.** Amer. Jour. Ophth., 1939, v. 22, Oct., pp. 1071-1082.

Silva, Linnen. **Pathogenesis of ocular complications in chickenpox.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 558-561.

The author has seen about fifty cases of ocular complications, chiefly corneal ulcers, in patients affected with chickenpox. Occasionally the corneal lesions were accompanied by iritis or iridocyclitis. The author believes that the virus responsible for the disease affects the ophthalmic branch of the trigeminus, which, thus irritated, is the cause of the ocular affection.
Ramon Castroviejo.

18

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Conde, Herminio. **Occupational ocular affections in the presence of the new legislation.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 541-549.

This is an analysis of the Brazilian legislation for compensation cases from the point of view of an ophthalmologist.
Ramon Castroviejo.

Currier, F. P. **Certain reading disabilities as related to speech.** Jour. Michigan State Med. Soc., 1938, v. 37, May, p. 414.

A discussion of some psychologic aspects of reading and speech difficulties, especially those of "mirror readers." Since most patients presenting these difficulties are left-handed, several suggestions are offered regarding the edu-

cation and training of left-handed children in the lower grades in school.

George A. Filmer.

Davidson, M. **The state labor-department ophthalmologist and his functions.** Industrial Med., 1939, v. 8, April, pp. 153-156.

The author is one of two full-time ophthalmologists attached to the Labor Department, Division of Workmen's Compensation of the State of New York, for the purpose of examining claimants for compensation. While eye injuries constitute only approximately 3 percent of all industrial injuries, the average cost per case (around $600.00) is about twice as much as for other injuries. The author outlines the sources of medicolegal controversies in eye cases and suggests a standard procedure of ocular examination for compensation cases. T. E. Sanders.

Hirschfelder, Max. **Trachoma in Illinois.** Illinois Med. Jour., 1939, v. 75, April, p. 368.

Old and new patients attending the Southern Illinois trachoma clinics were examined in August, 1938, and listed according to the stage of the trachomatous disease present. In general the system used in the listing followed the description of MacCallan. The figures in the statistical table show that the percentage of active, malignant trachoma in Southern Illinois is now relatively small (5 percent). Earlier recognition and treatment are important parts of the task of the Southern Illinois trachoma service whose aim it is not only to control, but to eliminate the disease. F. M. Crage.

Lordan, J. P. **Blind laws in California.** California and Western Med., 1939, v. 50, Feb., p. 114.

A statistical summary presented largely in tabular form showing number of blind individuals, cost to the state, age, sex, and race grouping, causes of blindness, and other items.

George A. Filmer.

Pfingst, O. A. **Development of ophthalmology in Kentucky.** Kentucky Med. Jour., 1938, v. 36, Dec., p. 561.

The article consists of short biographic sketches of pioneer ophthalmologists in Kentucky during the last half of the nineteenth century. Most of these men were known by the author, and his descriptions of their personal and professional characteristics are particularly interesting. (Illustrations.)

George A. Filmer.

Selling, L. S. **Abnormalities of the eye in traffic court cases.** Jour. Amer. Med. Assoc., 1939, v. 113, Sept. 9, p. 994.

This report deals with 716 persons who were given complete clinical examinations, 45 control subjects secured from the public at large, and a similar control group of minor violators consisting of 141 persons. Sensitivity to glare, visual fields, and color vision were tested. No definite conclusions as to glare sensitivity were reached, but apparently those with more pigmented retinas are less sensitive, and diet may be less important than is now thought. Visual-field impairment was seldom found and color blindness does not necessarily interfere with a person's ability to drive a motor car. The determination of the adequacy of the retinal function is, however, very important. (3 tables, discussion.)

George H. Stine.

Spinola, Colombo. **Under our law of industrial accidents, how much is one eye worth?** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 2, pp. 552-556.

The author discusses in detail the compensation to be paid, under Brazilian law, for the partial or total loss of one eye. Ramon Castroviejo.

Thacker, E. A. **A study of the professions testing ocular refraction.** Amer. Jour. Ophth., 1939, v. 22, Nov., pp. 1227-1239.

Vogelsang, K. **The ophthalmologist as pediatrist.** Klin. M. f. Augenh., 1939, v. 103, Aug., p. 235.

The activity of the ophthalmologist in the care of individuals between six and 18 years of age is discussed. Pathologic conditions include scrofulosis, parenchymatous keratitis, follicular conjunctivitis, vernal catarrh, diseases of the lens, juvenile relapsing hemorrhages, anomalies of refraction, strabismus, and juvenile glaucoma.

C. Zimmermann.

19

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPHTHALMOLOGY

Contino, Felippo. **The eye of Argyropelecus hemigymnus.** Graefe's Arch., 1939, v. 140, pt. 2, pp. 290-441.

Argyropelecus is a small deep-sea fish with elongated telescopic eyes. The author, after a study of over one hundred specimens of the embryonic, immature, and mature forms, describes the anatomy of the orbits and eyeballs, the histology of the globes, the embryology and development of the eyes, and optical investigations relating to refraction. The eye of this fish is highly myopic (about 64 diopters); and the far point in accommodation (through action of the retractor lentis) reaches over 40 mm. The writer believes it likely that the deep-sea fish use for their vision light derived from fluorescence. Charles A. Perera.

NEWS ITEMS

Edited by Dr. H. Rommel Hildreth

640 S. Kingshighway, Saint Louis

News items should reach the Editor by the twelfth of the month

Deaths

Dr. Max Halle, New York, died September 5, 1939, aged 66 years.

Dr. Arthur Godfrey Fort, Atlanta, Georgia, died September 15, 1939, aged 61 years.

Dr. James Edward Francis Cocan, Cleveland, Ohio, died September 22, 1939, aged 68 years.

Dr. Ashley Walker Morse, Butte, Montana, died October 12, 1939, aged 56 years.

Miscellaneous

The George Washington University School of Medicine, Washington, D.C., announces the Fourth Annual Intensive Postgraduate Course in Ophthalmology, March 25 to 30, 1940 (inclusive). Subjects by guest lecturers are: two lectures daily on motor anomalies of the eye illustrated with case demonstration, vascular changes in the ocular fundus, diseases of the optic nerve, ocular therapeutics, the surgical treatment of cataract, the ophthalmoscopic picture of retinal detachment and its interpretation, ocular syndromes, allergy in relation to ophthalmology, glaucoma, applied refraction, differential diagnosis of the phorias, sympathetic ophthalmia, industrial ophthalmology, plastic surgery of the eye, pathology of the eye, conjunctivitis, slitlamp findings of trauma to the eye and lens, color photograph clinic of rare and interesting fundi and anterior-eye conditions, and ophthalmic lenses. Also, there is a special practical course limited to 25 participants March 19 to 23, 1940 (inclusive)—ocular surgery, pathology, and orthoptics—given by the resident staff of the Department of Ophthalmology of the George Washington University School of Medicine and the Army Medical Museum. This includes surgery of the eye on cadaver and animal eyes, ocular pathology at the Army Medical Museum with material from the Registry of Ophthalmic Pathology of the American Academy of Ophthalmology and Otolaryngology, and a practical course in the technique of orthoptic training.

The National Society for the Prevention of Blindness, Inc. (incorporated in the state of New York) is rendering a service throughout the United States. It is a membership and nonprofit health organization, supported by voluntary contributions, membership dues, legacies, and bequests; it receives no Federal, state, or city aid, nor grants from any community chests. It is endorsed by the National Information Bureau, Inc., 215 Fourth Avenue, New York, New York, and is a member of the National Health Council, 50 West 50th Street, New York, New York.

This corporation is concerned with the control, and, where possible, the elimination of the causes of blindness, impaired vision, and eyestrain—not with activities on behalf of those already blind. In this respect it operates in a field peculiarly its own and performs a much needed service to society. Particular attention is given to: 1. Coöperating with the medical profession in devising measures and instituting procedures for the conservation of vision and the reduction of blindness. 2. Collaborating with those in industry who are striving to reduce ocular injuries and eyestrain. 3. Assisting nurses to become increasingly aware of their opportunities for conserving sight, and of the relationship between eye health and general health. 4. Demonstrating the value of trained medical social workers in eye hospitals and clinics and helping such workers to secure specialized training. 5. Coöperating with educational authorities in: a. Conserving the vision of school and college students. b. Establishing sight-saving classes for children whose vision is so defective that they cannot profitably use ordinary school equipment. c. Providing specialized training for teachers of sight-saving classes. d. Helping student-teachers secure better preparation for meeting the eye-health problems of school children. 6. Stressing the value of properly caring for the eyes of preschool children and demonstrating an approved method of testing their vision in order to discover those who will benefit from early treatment. 7. Furthering the universal use of prevention measures before and at birth to protect babies' eyes from infection. 8. Encouraging adequate prenatal care for every expectant mother, including a blood test and treatment when necessary as the means of preventing blindness from prenatal syphilis. 9. Furnishing information regarding the relationship between the conservation of vision and numerous environmental factors, including quality and intensity of illumination, size and style of type, quality of paper, and so forth. 10. Stimulating further investigation and study of the causes of blindness and impaired vision. 11. Counseling governmental and voluntary agencies working for the conservation of vision. 12. Serving as a clearing-house on all matters pertaining to the prevention of blindness and the conservation of vision; providing the public with information concerning the care and use of the eyes.

The nature of the society's work is such that its public usefulness can be materially enhanced by any increase in its resources. There are numerous fields in which it could be of great public benefit, but from which the society is now precluded, or in which its activity is limited, for lack of sufficient funds. Inquiries for further information welcomed.

Thousands of workers in New York State have been saved from blindness or serious damage to their eyes through a control of poisonous gases and chemicals in industry, it was reported by Dr. Leonard Greenburg, executive director of the Division of Industrial Hygiene, New York State Department of Labor, at the annual conference here of the National Society for the Prevention of Blindness. Dr. Greenburg revealed that his department has approved, during the past year, approximately 2,000 sets of engineering plans for the control of such toxic materials in New York factories. He pointed out that New York is the only state that exercises such complete control.

"Among the substances which were controlled and which produced damage to the eyes," said Dr. Greenburg, "are carbon tetrachloride, alcohol, carbon monoxide, carbon bisulphide, hydrogen sulphide, lead, and many others. These are only a few of the many poisons used in industry today which may cause blindness or damage to the eyes."

Dr. Greenburg also recommended a mandatory rule requiring the wearing of goggles by all workers engaged in occupations that present serious hazards to the eyes. "Is it not just as important to mandate the protection of eyes in hazardous occupations," he remarked, "as it is to promulgate codes requiring the use of guards on dangerous gears and punch presses?

"There appears to be no reason why the state should not take the same attitude with reference to the protection of eyes, by means of suitable guarding in the form of goggles, as the state takes with reference to the protection of the respiratory organs by means of masks and respirators or by the introduction of control devices for the removal of dusts and toxic materials. The chief role of the state would then consist in the promulgation of suitable codes having the full effect of law, and the enforcement of these in industry.

"There is no doubt that goggles are necessary whenever an eye accident occurs, but who can say just when and where an eye accident may happen? In fact, by definition, an accident is something unexpected; hence, it would be impossible to predict, in most cases, just when goggles should be employed. It seems obvious from this that from the point of view of industry the goggle campaign should have as its end the compulsory use of goggles throughout the full working period of all employees. Only by such a compulsory campaign may the employer discharge his duty and prevent accidents practically 100 percent of the time.

"In spite of the joint efforts of labor, industry, voluntary and official agencies, a tremendous toll of needless eye damage still continues to be inflicted on the workers in the factories, day in and day out, year after year, in New York State and throughout the United States. The challenge which we must now meet is—How may we proceed to reduce this tremendous loss to some irreducible level? The eye accidents in industry have a cost far greater than the direct monetary cost. The accumulated social cost of such accidents is tremendous, and the cost to the individual worker in pain and both physical and psychic suffering is devastating in many cases. The total cost of eye injuries cannot be evaluated with any degree of accuracy."

The University of Buffalo awards annually a gold medal for work in an ophthalmologic subject. For details write: H. W. Cowper, D.M., 543 Franklin Street, Buffalo, New York.

Training for orthoptic technicians. The Staff of the Illinois Eye and Ear Infirmary announce a six-month course for the training of orthoptic technicians to be given at the infirmary beginning January, 1940. Students must be high-school graduates, must pass a normal eye test, and must have a letter of sponsorship from an oculist certificated by the American Board of Ophthalmology. Price of course $150.00, half of which is payable on registration. Address inquiries to Dean of Instruction, Illinois Eye and Ear Infirmary, 904 West Adams Street, Chicago, Illinois.

The Ninth Annual Mid-Winter Course will be given by the Research Study Club of Los Angeles from January 15th to January 26th, inclusive. It will embrace both ophthalmology and otolaryngology. The teaching staff in ophthalmology will include: Dr. Albert D. Ruedemann, Dr. Algernon B. Reese, Dr. Meyer Wiener, Dr. Edward Jackson, Dr. Russell L. Stimson.

The nursing staff of the Department of Ophthalmology, Long Island College Hospital, presented a special program on November 2d, in Hoagland Auditorium. This program was so interesting and so well attended that it should serve as a model for those interested in the general problem of the prevention of blindness.

Mr. Elliott S. Humphrey, vice-president of "The Seeing Eye," Morristown, New Jersey, spoke on the Education of the guide dog, the value of them to the blind, the inspiration and independence that has been theirs since this opportunity has been made possible. His talk was illustrated with moving pictures: "Dogs against darkness."

"Cataracts, types, etiology, and treatment"

was outlined by Dr. Frank E. Mallon. Colored moving pictures of the Moehle cataract operation were shown and elaborated upon by Dr. Walter Moehle. The various eye instruments, their delicacies, importance in handling, and care to be used was explained by Dr. George Freiman, and lantern slides were shown as well as several boxes of knives passed through the audience for close scrutiny.

Dr. Henry Abbott enumerated the necessities and preparations required prior to admission and operation and presented the patient for hospitalization. A short nursing pantomime entitled "Seeing through" was given by the ophthalmological nurses. This depicted nursing care from the admission of the patient through the entire hospital stay to the day of discharge.

The new Altherm Eye Pad was prepared and passed through the audience that each one present might test the benefit of this modern procedure of applying heat to the eye. Literature was available for all present. The individual dressing box, special eye cart, and many other ophthalmological literature textbooks, instruments, dressings, and so forth, were on exhibit, and nurses were at hand to explain their use and importance.

The International Association for the Prevention of Blindness is offering an honorarium of $1,000 to further research in ophthalmology. Papers are to include anything definitely relative to simple non-inflammatory glaucoma, any responsible research worker being eligible. The secretary of the International Association for the Prevention of Blindness may be reached at 66 Boulevard Saint-Michel, Paris, for details.

Societies

The eye section of the Philadelphia County Medical Society presented the following program on December 7, 1939: Clinic case, Wills Hospital, Dr. Miles O'Brien; Clinic case, Temple University Hospital, Dr. M. Wilson Snyder; Neurologic type of sympathetic ophthalmia with recovery in the sympathizing eye, Dr. Edmund B. Spaeth; Cavernous sinus thrombosis as it applies to facial surgery, Dr. William Bates.

The Cleveland Ophthalmological Club, in conjunction with the Frank E. Bunts Institute, sponsored a postgraduate course in ophthalmology on December 4, 5, and 6, 1939, at the Cleveland Clinic.

The Ophthalmological Congress meets at Oxford, July 4, 5, and 6, 1940.

The trustees of the Association for Research in Ophthalmology announce that at the 1940 meeting a cash prize of $100.00 will be offered for the paper which in their judgment shows most originality and exemplifies best the spirit of research in ophthalmology.

Personals

The Leslie Dana Gold Medal awarded annually for "outstanding achievements in the prevention of blindness and the conservation of vision" was presented to Dr. Edward C. Ellett, of Memphis, Tennessee, at a dinner in his honor in Saint Louis, Saturday evening, October 14th. The presentation was made by Dr. Edward Jackson, of Denver, Colorado, who was the first recipient of the Leslie Dana Medal, in 1925. Another speaker was Lewis H. Carris, of New York City, general director of the National Society for the Prevention of Blindness.

Dr. Ellett, who is 69 years old, has practiced ophthalmology in Memphis since 1893, and he was professor of diseases of the eye at the University of Tennessee from 1906 to 1922. He is former chairman of the section on ophthalmology of the American Medical Association, former president of the American Academy of Ophthalmology and Otolaryngology, and former president of the American Ophthalmological Society.

The inscription on the 1939 medal refers to Dr. Ellett as an "Inspiring Teacher, Skilled Surgeon, Understanding and Sympathetic Clinician and Friend."

The conditions of the Leslie Dana Medal award set forth that it is to be made for "long meritorious service in the conservation of vision, in the prevention and cure of diseases dangerous to eyesight; research and instruction in ophthalmology and allied subjects; social service for the control of eye diseases; and special discoveries in the domain of general science or medicine of exceptional importance in conservation of vision."

At a dinner meeting of the Cleveland Ophthalmological Club, held September 26th, Dr. Georgiana Theobald of Oak Park, Illinois, was the speaker of the evening. Dr. Theobald gave a very comprehensive, instructive, and interesting paper on "Pathological end results of glaucoma surgery."

Dr. Vincent J. Kelly of Chicago, Illinois, was recently appointed senior intern on the ophthalmological service of the University Hospitals of Cleveland. Dr. Irwin H. Stolzer of Cleveland, Ohio, was appointed junior intern.

At a recent meeting of the Ohio State Safety Council, held in Cleveland, Dr. Paul Motto spoke on the Relationship between defective vision and automobile accidents.

Mr. Edward Bacon has been appointed honorary assistant ophthalmic surgeon to the Southampton Free Eye Hospital.